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A STOCHASTIC STUDY OF THE LIFE TABLE AND ITS APPLICATIONS: II. SAMPLE VARIANCE OF THE OBSERVED EXPECTATION OF LIFE AND OTHER BIOMETRIC FUNCTIONS[1]

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IN the first paper of this series we were concerned with the theoretical probability distributions of biometric functions in the life table (Chiang, in press A). It was shown that (1) the numbers of survivors to the ages specified by the life table form a binomial random vector with a positive covariance between any two of the components; (2) the numbers of deaths in the specified age intervals have a multinomial distribution; (3) the covariance between the proportions of deaths (or survivors) in any two age intervals is zero, providing these intervals do

[1] Parts of this paper were presented at the joint meeting of the American Statistical Association and the Biometric Society, ENAR, in Atlantic City, September 13, 1957, under the title, "An application of stochastic processes to the life table and standard error of the age adjusted rate" (Chiang, 1958). The work was completed during the tenure of a Special Fellowship from the National Heart Institute, U. S. Public Health Service.

not overlap; and (4) the distribution of the observed expectation of life at a given age is approximately normal if the number of survivors to that age is sufficiently large. Each of these functions was found to be an unbiased estimator of the corresponding unknown true value, and formulas were derived for their variance and covariance. Application of these theoretical results to actual problems of statistical inference necessitates formulas for the corresponding sample variance and covariance. Such formulas are presented in this paper.

Although the theoretical probability distributions hold for both cohort (generation) life tables and current life tables, it is important in deriving formulas for their sample variances to recall that the basic random variables are different in the two cases. A cohort life table records the actual mortality experience of a given group of individuals from the time of birth to the death of the last member of the group. The basic random variables here are the number of survivors and the number of deaths, quantities that are directly observable in the experience of the population. The current life table is constructed from the proportion of deaths as determined by the age specific central death rate observed at one point in time. Other biometric functions are random variables only because they are functions of this one proportion. Thus, in the current life table the basic random variable is the proportion of deaths. Formulas for these two types of tables will be presented separately.

The emphasis in this presentation is placed on the sample variance of the observed expectation of life. Formulas for the sample variance and covariance of the number of survivors, the number of deaths, and the proportion of deaths are included for the sake of completeness, although the derivations are rather obvious. Because of their related interest, formulas are also presented for the sample variance of the age specific death rate and for the x_i-year survival rate.

Symbols used in this paper, as in the previous paper (Chiang, in press A), deviate slightly from the conventional ones, and are defined in table 1. A symbol $\wedge$ is added to q_i and e_i to distinguish the sample estimators from their unknown true values. Thus $\hat{q}_i$ denotes the observed proportion of individuals dying in the age interval (x_i, x_{i+1}), and q_i the true probability that an individual alive at age x_i will die in the interval (x_i, x_{i+1}). The symbols $\hat{e}_i$ and e_i denote, respectively, the observed and the unknown true expectation of life at age x_i. The difference $x_{i+1} - x_i = n_i$ is the length of the interval. The age x_0 may be taken as 0, the time of birth, and the symbol x_w is used to denote the age at the beginning of the last age interval after which no survivors are left.

The term "estimated probability of death" will also be used instead of "proportion of deaths" for $\hat{q}_i$ in the discussion on the current life table.

TABLE 1

The Life Table

AGE INTERVAL (YEARS)	NUMBER OF SURVIVORS TO AGE x_i	PROPORTION OF DEATHS WITHIN AGE INTERVAL (x_i, x_{i+1})	NUMBER OF DEATHS WITHIN AGE INTERVAL (x_i, x_{i+1})	NUMBER OF YEARS LIVED WITHIN AGE INTERVAL (x_i, x_{i+1})	TOTAL NO. OF YEARS REMAINING TO SURVIVORS AT AGE x_i	OBSERVED EXPECTATION OF LIFE AT AGE x_i
x_i to x_{i+1}	l_i	$\hat{q}_i$	d_i	L_i	T_i	$\mathring{e}_i$
x_0 to x_1	l_0	$\hat{q}_0$	d_0	L_0	T_0	$\mathring{e}_0$
.	.	.	.	.	.	.
.	.	.	.	.	.	.
x_w to x_{w+1}	l_w	$\hat{q}_w$	d_w	L_w	T_w	$\mathring{e}_w$

COHORT LIFE TABLE

1. *Number of survivors and number of deaths and the corresponding proportions.* The sample variance and covariance of these biometric functions can be obtained simply by replacing the unknown quantities with the observed values in the respective formulas developed in the first paper (Chiang, in press A). Thus, the sample variance of l_i, the number of survivors at the age x_i, will have the form

$$S_{l_i}^2 = l_0 \hat{p}_{0i}(1 - \hat{p}_{0i}), \quad \text{for } i = 1, \cdots, w, \tag{1}$$

where

$$\hat{p}_{0i} = l_i / l_0,$$

and the sample covariance

$$S_{l_i, l_j} = l_0 \hat{p}_{0j}(1 - \hat{p}_{0i}), \quad \text{for } i \leqq j; i, j = 1, 2, \cdots, w. \tag{2}$$

The distribution of the numbers of deaths, d_i, occurring in the intervals (x_i, x_{i+1}), for $i = 1, \cdots, w$, is multinomial and has the sample variance

$$S_{d_i}^2 = (1/l_0) d_i (l_0 - d_i), \quad \text{for } i = 1, \cdots, w, \tag{3}$$

and the sample covariance

$$S_{d_i, d_j} = -(1/l_0) d_i d_j, \quad \text{for } i \neq j; i, j = 1, \cdots, w. \tag{4}$$

The proportion of deaths, $\hat{q}_i$, and the proportion of survivors, $\hat{p}_i$ are complementary and have the same sample variance,

$$S_{\hat{q}_i}^2 = S_{\hat{p}_i}^2 = (1/l_i)\hat{p}_i\hat{q}_i, \quad \text{for } i = 1, \cdots, w. \tag{5}$$

The covariance between proportions of deaths (or survivors) occurring in two intervals was shown to be zero, providing these intervals do not overlap; otherwise, the covariance is positive. For example, the sample covariance between the proportions of survivors in two age intervals (x_α, x_i) and (x_α, x_j), both beginning at age x_α, has the formula

$$S_{\hat{p}_{\alpha i}, \hat{p}_{\alpha j}} = (1/l_\alpha)\hat{p}_{\alpha j}(1 - \hat{p}_{\alpha i}), \quad \text{for } \alpha < i \leqq j; \alpha, i, j = 0, 1, \cdots, w. \tag{6}$$

2. *Observed expectation of life, $\hat{e}_\alpha$.* The observed expectation of life at a particular age, say x_α, is the average future lifetime of the l_α individuals alive at age x_α. The future lifetime of an individual alive at age x_α and dying in some interval (x_i, x_{i+1}) subsequent to x_α will, of course, be $x_i - x_\alpha$ plus some fractional part of n_i. If we assume a value for this fraction such that each of these d_i individuals who die lives an average of $a_i n_i$ years in the interval (x_i, x_{i+1}), then for each of these individuals the future lifetime beyond age x_α is

$$Y_\alpha = x_i + a_i n_i - x_\alpha, \quad \text{for } i = \alpha, \alpha + 1, \cdots, w. \tag{7}$$

Since the life table covers the entire life span of the individuals under consideration,

$$\sum_{i=\alpha}^{w} d_i = l_\alpha$$

and hence the average future lifetime of the l_α individuals is

$$\hat{e}_\alpha = (1/l_\alpha) \sum_{i=\alpha}^{w} (x_i + a_i n_i - x_\alpha) d_i, \quad \text{for } \alpha = 0, 1, \cdots, w. \tag{8}$$

By substituting $l_i - l_{i+1} = d_i$ and

$$c_i = (1 - a_{i-1}) n_{i-1} + a_i n_i \tag{9}$$

we can rewrite formula (8) as

$$\hat{e}_\alpha = a_\alpha n_\alpha + \sum_{i=\alpha+1}^{w} c_i \hat{p}_{\alpha i}, \quad \text{for } \alpha = 0, 1, \cdots, w, \tag{10}$$

which is now a linear function of the proportions $\hat{p}_{\alpha i}$. Therefore, the sample variance of $\hat{e}_\alpha$ is

$$S_{\hat{e}_\alpha}^2 = \sum_{i=\alpha+1}^{w} c_i^2 \, S_{\hat{p}_{\alpha i}}^2 + 2 \sum_{i=\alpha+1}^{w-1} \sum_{j=i+1}^{w} c_i c_j S_{\hat{p}_{\alpha i}, \hat{p}_{\alpha j}}, \quad \text{for } \alpha = 0, \cdots, w. \tag{11}$$

Substituting formulas (5) and (6) for the sample variance and covariance of the proportions of survivors gives

$$S_{\hat{e}_\alpha}^2 = \sum_{i=\alpha+1}^{w} c_i^2 (1/l_\alpha)\hat{p}_{\alpha i}(1-\hat{p}_{\alpha i}) + 2\sum_{i=\alpha+1}^{w-1}\sum_{j=i+1}^{w} c_i c_j (1/l_\alpha)\hat{p}_{\alpha j}(1-\hat{p}_{\alpha i}). \quad (12)$$

Following the same lines of simplification as in (Chiang, in press A), formula (12) may be reduced to

$$S_{\hat{e}_\alpha}^2 = \sum_{i=\alpha}^{w-1} \hat{p}_{\alpha i}^2 [\hat{e}_{i+1} + (1-a_i)n_i]^2 S_{\hat{q}_i}^2, \quad \text{for } \alpha = 0, \cdots, w-1, \quad (13)$$

where the sample variance of $\hat{q}_i$ is given by (5).

The formula for the sample variance of $\hat{e}_\alpha$ can also be derived from another approach. We notice from (8) that the observed expectation of life at age x_α is the sample mean of Y_α, and that their sample variances have the relation

$$S_{\hat{e}_\alpha}^2 = (1/l_\alpha) S_{Y_\alpha}^2. \quad (14)$$

When the life table, from age x_α on, is visualized as a frequency table of l_α future lifetimes with a value of $x_i + a_i n_i - x_\alpha$ in the interval (x_i, x_{i+1}) and a corresponding frequency of d_i, the sample variance of the future lifetime may be computed from the formula

$$\begin{aligned} S_{Y_\alpha}^2 &= (1/l_\alpha) \sum_{i=\alpha}^{w} [(x_i + a_i n_i - x_\alpha) - \hat{e}_\alpha]^2 d_i \\ &= (1/l_\alpha)[\sum_{i=\alpha}^{w} (x_i + a_i n_i)^2 d_i - l_\alpha(\hat{e}_\alpha + x_\alpha)^2]; \end{aligned} \quad (15)$$

consequently the sample variance of the observed expectation of life at age x_α is simply

$$S_{\hat{e}_\alpha}^2 = (1/l_\alpha^2)[\sum_{i=\alpha}^{w} (x_i + a_i n_i)^2 d_i - l_\alpha(\hat{e}_\alpha + x_\alpha)^2], \quad \text{for } \alpha = 0, \cdots, w. \quad (16)$$

Formulas (12), (13), and (16) are of course identical, although perhaps not equally convenient for computation in all cases. Proof of the identity of formulas (13) and (16) is given in the Appendix.

If the distribution of deaths within each age interval is such that $a_i = \frac{1}{2}$, so that $c_i = 1$, then formulas (12), (13), and (16) reduce, respectively, to

$$S_{\hat{e}_\alpha}^2 = (1/l_\alpha)[\sum_{i=\alpha+1}^{w} \hat{p}_{\alpha i}(1-\hat{p}_{\alpha i}) + 2\sum_{i=\alpha+1}^{w-1}\sum_{j=i+1}^{w} \hat{p}_{\alpha j}(1-\hat{p}_{\alpha i})], \quad (17)$$

$$S_{\hat{e}_\alpha}^2 = \sum_{i=\alpha}^{w-1} \hat{p}_{\alpha i}^2 [\hat{e}_{i+1} + n_i/2]^2 S_{\hat{q}_i}^2, \tag{18}$$

and

$$S_{\hat{e}_\alpha}^2 = (1/l_\alpha^2)[\sum_{i=\alpha}^{w} (x_i + n_i/2)^2 d_i - l_\alpha(\hat{e}_\alpha + x_\alpha)^2], \quad \text{for } \alpha = 0, \cdots, w. \tag{19}$$

If further the age interval is of a constant length n, formula (18) may be simplified to (Chiang, in press A)

$$S_{\hat{e}_\alpha}^2 = (1/l_\alpha^2)[2n \sum_{i=\alpha}^{w} l_i \hat{e}_i - l_\alpha(\hat{e}_\alpha + n/2)^2]$$

$$= (1/l_\alpha^2)[2n \sum_{i=\alpha}^{w} T_i - l_\alpha(\hat{e}_\alpha + n/2)^2], \quad \text{for } \alpha = 0, \cdots, w, \tag{20}$$

where T_i is the total number of years remaining to survivors at age x_i; and formula (19) becomes

$$S_{\hat{e}_\alpha}^2 = (1/l_\alpha^2)[\sum_{i=\alpha}^{w} x_i^2 d_i - l_\alpha(\hat{e}_\alpha + x_\alpha - n/2)^2], \quad \text{for } \alpha = 0, \cdots, w. \tag{21}$$

Example 1: In an experimental study of the longevity of *Drosophila melanogaster*, 270 male adults and 275 female adults were raised at a larval density of 20 per vial and followed until the death of the last member in each group. Their mortality experience is recorded in the following life table (Miller and Thomas, 1958).

Here the age intervals are a constant length of $n = 5$ days, and deaths are assumed to have occurred uniformly within each interval; thus the sample variance of the observed expectation of life may be computed with either formula (20) or formula (21). Particularly if the column headed T_i has been included in the life table, formula (20) is more conveniently used. The sample standard error (square root of the sample variance) of the observed expectation of life at each age for each sex is entered in columns (6) and (11).

An exact statistical test for the difference between expectations of life of two population must take into account the form of the force of mortality, and no general solution is yet available. If the sample size is large enough, however, the normal test may be used (Chiang, in press A). For example, in the above study the normal approximation is appropriate for testing the hypothesis concerning the expectation of life at age zero,

TABLE 2

Life table for adult Drosophila melanogaster raised at a larval density of 20 per vial

$x_i - x_{i+1}$ (DAYS)	MALES					FEMALES				
	l_i	d_i	$1000\hat{q}_i$	$\hat{e}_i$	$S_{\hat{e}_i}$	l_i	d_i	$1000\hat{q}_i$	$\hat{e}_i$	$S_{\hat{e}_i}$
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
0– 5	270	2	7.41	43.19	.698	275	4	14.55	37.47	.676
5–10	268	4	14.93	38.49	.671	271	7	25.83	32.98	.636
10–15	264	3	11.36	34.03	.624	264	3	11.36	28.79	.575
15–20	261	7	26.82	29.40	.594	261	7	26.82	24.09	.555
20–25	254	3	11.81	25.14	.540	254	13	51.18	19.69	.522
25–30	251	3	11.95	20.41	.524	241	22	91.29	15.61	.484
30–35	248	16	64.52	15.63	.514	219	31	141.55	11.93	.443
35–40	232	66	284.48	11.53	.498	188	68	361.70	8.48	.419
40–45	166	36	216.87	10.12	.460	120	51	425.00	6.88	.406
45–50	130	54	415.38	7.23	.433	69	38	550.72	5.11	.378
50–55	76	42	552.63	5.59	.436	31	26	838.71	3.31	.330
55–60	34	21	617.65	4.41	.417	5	5	1000.00	2.50	—
60 and over	13	13	1000.00	2.50	—					

where the sample sizes are 270 for males and 275 for females. Thus one may conclude from the computations

$$\frac{\hat{e}_0(M) - \hat{e}_0(F)}{\text{S. E.(diff)}} = \frac{43.19 - 37.47}{\sqrt{\frac{270(.698)^2 + 275(.676)^2}{270 + 275 - 2}}} = 8.31$$

that under the experimental conditions in question the male *Drosophila* has a greater longevity than the female. But in the case of $\hat{e}_{55}$, with sample sizes of 34 for males and 5 for females, the assumption of normality will be quite strong.

CURRENT LIFE TABLE

1. *Estimated probability of death, $\hat{q}_i$.* Since the basic random variable in the current life table is $\hat{q}_i$, the estimated probability of death in each age interval, the sample variance of other biometric functions should be expressed in terms of the sample variance of this one variable. But $\hat{q}_i$ is obtained, in turn, from the age specific death rate of the population in question, and hence it may be desirable to express its sample variance as a function of the latter.

The age specific death rate is defined as

$$M_i = D_i/P_i \tag{22}$$

where D_i is the number of deaths occurring to persons in age interval (x_i, x_{i+1}) during a calendar year and P_i is the corresponding mid-year population. The estimated probability of death, on the other hand, may be written as

$$\hat{q}_i = D_i/N_i, \tag{23}$$

where N_i is a hypothetical population size at the beginning of the age interval (x_i, x_{i+1}) in which D_i deaths will occur, if the force of mortality prevailing in the population acts on each of the N_i individuals. The sample variance of $\hat{q}_i$ has the usual form

$$S_{\hat{q}_i}^2 = \hat{q}_i(1-\hat{q}_i)/N_i. \tag{24}$$

Although N_i is unknown but it may be estimated from (23) to give the required formula

$$S_{\hat{q}_i}^2 = \hat{q}_i^2(1-\hat{q}_i)/D_i. \tag{25}$$

Since D_i is usually given, formula (25) can easily be computed.

If one wishes to express the sample variance of $\hat{q}_i$ in terms of the age specific death rate, then it is necessary to know the relationship between $\hat{q}_i$ and M_i used in the construction of the life table. Various methods have been suggested in the literature [See, for example, (Greville, 1943) and (Reed and Merrell, 1939)], and in general it is assumed that $\hat{q}_i$ is a function of M_i,

$$\hat{q}_i = f_i(M_i). \tag{26}$$

Substitution of (26) in (25) gives

$$\begin{aligned} S_{\hat{q}_i}^2 &= \frac{[f_i(M_i)]^2[1-f_i(M_i)]}{D_i} \\ &= \frac{[f_i(M_i)]^2[1-f_i(M_i)]}{P_iM_i}. \end{aligned} \tag{27}$$

Consider, for example, the function

$$\hat{q}_i = \frac{n_iM_i}{1+(1-a_i)n_iM_i} \tag{28}$$

often used to compute $\hat{q}_i$ from M_i. Here again a_in_i may be interpreted as the average number of years lived in the interval (x_i, x_{i+1}) by an individual who dies in that interval. Direct computation from (27) gives

$$S_{\hat{q}_i}^2 = \frac{n_i^2 M_i(1 - a_i n_i M_i)}{P_i[1 + (1 - a_i) n_i M_i]^3}. \tag{29}$$

The sample variance of $\hat{q}_i$ as given by (29) can never be negative. Clearly the denominator is always positive, since a_i is a fractional number. For the numerator we observe that, being an estimator of a probability, the function given in (28) can never be greater than unity, and this implies that $1 - a_i n_i M_i$ can never be negative, proving the assertion. The sample variance will be zero if and only if either $M_i = 0$ or $\hat{q}_i = 1$, two rather trivial cases.

If the distribution of deaths in the interval (x_i, x_{i+1}) is such that $a_i = \frac{1}{2}$, then formulas (28) and (29) reduce, respectively, to

$$\hat{q}_i = \frac{2 n_i M_i}{2 + n_i M_i}, \tag{30}$$

and

$$S_{\hat{q}_i}^2 = \frac{4 n_i^2 M_i(2 - n_i M_i)}{P_i(2 + n_i M_i)^3}. \tag{31}$$

Remark 1: While it is not the purpose of this paper to discuss the sample variance of an age specific death rate, for the convenience of a later discussion, it may be mentioned that the sample variance of M_i corresponding to formula (28) is given by

$$S_{M_i}^2 = \frac{M_i(1 - a_i n_i M_i)}{P_i[1 + (1 - a_i) n_i M_i]}. \tag{32}$$

A detailed study on the sample variance of a death rate may be found in Chiang (in press B).

2. *x_i-year survival rate, $\hat{p}_{0i}$.* In the current life table the number of deaths and the number of survivors at any age will depend upon l_0, the arbitrary radix chosen for convenience of computation. Thus the figures in these columns are not meaningful by themselves. The variance of l_i, however, is of interest in that it can be used to develop the sample variance of the popular 'x_i-year survival rate,'

$$\hat{p}_{0i} = l_i / l_0, \quad \text{for } i = 0, 1, \cdots, w. \tag{33}$$

To derive the formula for the sample variance of l_i, we first write

$$\begin{aligned} l_i &= l_0 \hat{p}_{0i} \\ &= l_0 \hat{p}_0 \hat{p}_1 \cdots \hat{p}_{i-1}, \end{aligned} \tag{34}$$

and then take the derivatives at the point $(\hat{p}_0, \hat{p}_1, \cdots, \hat{p}_{i-1})$

$$(\partial/\partial\hat{p}_h)l_i = l_0\hat{p}_h^{-1}\,\hat{p}_{0i}, \qquad \text{for } 0 \leqq h < i;$$
$$= 0, \qquad \text{for } h \geqq i.$$

Since the covariance between proportions of survivors in different age intervals is zero, the sample variance of l_i is given by

$$S_{l_i}^2 = l_0^2\hat{p}_{0i}^2 \sum_{h=0}^{i-1} \hat{p}_h^{-2} S_{\hat{p}_h}^2, \qquad \text{for } i = 0, 1, \cdots, w; \tag{35}$$

and the sample variance of $\hat{p}_{0i}$ by

$$S_{\hat{p}_{0i}}^2 = \hat{p}_{0i}^2 \sum_{h=0}^{i-1} \hat{p}_h^{-2} S_{\hat{p}_h}^2, \qquad \text{for } i = 0, 1, \cdots, w. \tag{36}$$

Here the sample variance of $\hat{p}_h$ is of course the same as that of $\hat{q}_h$ and may be computed from (25).[2]

3. *Observed expectation of life, $\hat{e}_\alpha$.* In Chiang (in press A) a formula for the variance of the observed expectation of life was derived on the basis of the observation that (1) $\hat{e}_\alpha$ may be expressed as a linear function of $\hat{p}_{\alpha i}$ [cf. eq. (10)] which, in turn, is a function of the proportion of survivors ($\hat{p}_h$); and (2) the covariance between any two of these proportions is zero. Consequently,

$$\sigma_{\hat{e}_\alpha}^2 = \sum_{i=\alpha}^{w-1} \{(\partial/\partial\hat{p}_i)\hat{e}_\alpha\}^2 \sigma_{\hat{q}_i}^2, \tag{37}$$

which, after simplication, becomes

$$\sigma_{\hat{e}_\alpha}^2 = \sum_{i=\alpha}^{w-1} p_{\alpha i}^2[e_{i+1} + (1 - a_i)n_i]^2 \sigma_{\hat{q}_i}^2, \quad \text{for } \alpha = 0, \cdots, w-1. \tag{38}$$

Substituting the observed values for the unknown quantities in (38) gives the sample variance of $\hat{e}_\alpha$,

$$S_{\hat{e}_\alpha}^2 = \sum_{i=\alpha}^{w-1} \hat{p}_{\alpha i}^2[\hat{e}_{i+1} + (1 - a_i)n_i]^2 S_{\hat{q}_i}^2, \quad \text{for } \alpha = 0, \cdots, w-1, \tag{39}$$

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[2] Formula (36) was first suggested by Greenwood (1926) as an approximation to his formula

$$\sigma_{\hat{p}_{0i}}^2 = p_{0i}^2\Big[\prod_{h=0}^{i-1}(1 + p_h^{-2}\sigma_{\hat{q}_h}^2) - 1\Big],$$

and has since been mentioned in papers by others (Elveback, 1958, Irwin, 1949, and Kaplan and Meier, 1958). The underlying assumption of Greenwood's formula is that the estimated probabilities, $\hat{p}_h$, are independently distributed. This assumption however is false (Chiang, unpublished).

where the sample variance of $\hat{q}_i$ is given in (25) and the constant a_i may be estimated from

$$a_i = \frac{L_i - n_i l_{i+1}}{n_i d_i}. \tag{40}$$

When the distribution of deaths in each interval is such that $a_i = \frac{1}{2}$, we have

$$S_{\hat{e}_\alpha}{}^2 = \sum_{i=\alpha}^{w-1} \hat{p}_{\alpha i}{}^2 [\hat{e}_{i+1} + \tfrac{1}{2} n_i]^2 S_{\hat{q}_i}{}^2, \quad \text{for } \alpha = 0, \cdots, w-1. \tag{41}$$

Remark 2: In deriving formulas for the variance of the observed expectation of life, it was assumed that $a_i n_i$ and consequently c_i are constant and that they are not subject to random variation. In the actual practice of life table construction, however, these constants are either arbitrarily assumed (*e. g.*, $a_i = \frac{1}{2}$), or are estimated on the basis of a large sample (as in the Reed and Merrell life table). It is impossible in the former case to assess their random variation, while in the latter their contribution to the total sample variation is negligible in comparison with that of $\hat{q}_i$, or M_i. The constant $a_w n_w$, the average number of years lived in the final age interval in a life table, presents a special case deserving clarification. Clearly, $a_w n_w = \hat{e}_w$ is the observed expectation of life at the beginning of the final age interval and is subject to sample variation; therefore the variance of $a_w n_w$ should not be neglected. For the practical importance, we consider the following two cases:

Case 1. The observed expectation of life $\hat{e}_w$ is computed on the basis of the assumption of a stationary population (Doering and Forbes, 1938) and is estimated from the equation

$$\hat{e}_w = 1/M_w, \tag{42}$$

where M_w is the corresponding age specific death rate. In such a case, the information is inadequate for evaluating a sample variation and thus the sample variance of $\hat{e}_w$ becomes zero; that is

$$S_{a_w n_w}{}^2 = S_{\hat{e}_w}{}^2 = 0. \tag{43}$$

For proof, we use (42) and write

$$S_{\hat{e}_w}{}^2 = [1/M_w]^4 S_{M_w}{}^2, \tag{44}$$

where the sample variance of the age specific death rate is obtained from (32),

$$S_{M_w}{}^2 = \frac{M_w (1 - a_w n_w M_w)}{P_w [1 + (1 - a_w) n_w M_w]}.$$

Since (42) implies

$$1 - a_w n_w M_w = 1 - \hat{e}_w M_w = 0,$$

the sample variance of M_w is zero, proving (43). Therefore, formulas (39) and (41) may be used without modification (See also: Chiang, in press B).

TABLE 3

Number of deaths, mid-year population, age specific death rate, and life table for white males, United States, 1955

AGE (YEARS) $x_i - x_{i+1}$	NUMBER OF DEATHS D_i	MID-YEAR POPULATION P_i	AGE SPECIFIC DEATH RATE PER 1000 1000 M_i	LIFE TABLE VALUES			
				l_i	$\hat{q}_i$	L_i	$\mathring{e}_i$
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
0– 1	47,457	1,595,000	29.8	100,000	.0267	97,609	67.3
1– 5	7,128	6,486,000	1.1	97,325	.0043	388,308	68.2
5–10	4,235	7,658,000	0.55	96,906	.0027	483,827	64.5
10–15	3,417	5,982,000	0.57	96,641	.0029	482,560	59.6
15–20	6,236	4,971,000	1.3	96,360	.0064	480,369	54.8
20–25	7,852	4,775,000	1.6	95,743	.0089	476,612	50.1
25–30	8,064	5,200,000	1.5	94,891	.0080	472,573	45.6
30–35	9,485	5,456,000	1.7	94,134	.0088	468,696	40.9
35–40	13,315	5,154,000	2.6	93,307	.0130	463,711	36.3
40–45	20,599	4,959,000	4.2	92,092	.0207	455,164	31.7
45–50	31,834	4,517,000	7.0	90,183	.0348	443,605	27.3
50–55	44,971	3,941,000	11.4	87,044	.0558	423,844	23.2
55–60	61,522	3,517,000	17.5	82,188	.0841	394,541	19.4
60–65	81,456	3,012,000	27.0	75,276	.1272	353,355	16.0
65–70	98,764	2,402,000	41.1	65,704	.1876	298,594	12.9
70–75	102,340	1,760,000	58.1	53,375	.2555	233,363	10.3
75–80	94,110	1,067,000	88.2	39,735	.3615	162,448	8.0
80–85	72,443	541,000	133.9	25,372	.4988	93,880	6.1
85 and over	58,987	294,000	200.6	12,717	1.0000	60,874	4.8

Case 2. For a small geographic area, or for other subpopulation where the number of individuals is small, mortality data of old age group are subject to a large random variation. The observed expectation of life at the beginning of the last age interval may be obtained through a special study, or replaced with that of the general population, or else it is estimated by special devices (Chiang, in press C). In any event,

if it is possible to evaluate the random variation, the variance of $\hat{e}_w$ should be taken into account in the study of the sample variance of $\hat{e}_\alpha$; and formulas (39) and (41) should be replaced, respectively, by

$$S_{\hat{e}_\alpha}{}^2 = \sum_{i=\alpha}^{w-1} \hat{p}_{\alpha i}{}^2 [\hat{e}_{i+1} + (1 - a_i) n_i]^2 \, S_{\hat{q}_i}{}^2 + \hat{p}_{\alpha w}{}^2 S_{\hat{e}_w}{}^2, \tag{45}$$

and

$$S_{\hat{e}_\alpha}{}^2 = \sum_{i=\alpha}^{w-1} \hat{p}_{\alpha i}{}^2 [\hat{e}_{i+1} + n_i/2]^2 \, S_{\hat{q}_i}{}^2 + \hat{p}_{\alpha w}{}^2 S_{\hat{e}_w}{}^2. \tag{46}$$

Example 2: United States mortality statistics will be used to illustrate the determination of the standard error of the expectation of life

TABLE 4

Computation of the sample variance of the observed expectation of life for white males, United States, 1955

AGE $x_i - x_{i+1}$ (1)	Sample variance of $\hat{q}_i$: $10^8 S_{\hat{q}_i}{}^2$ (2)	n_i (3)	a_i (4)	$10^{-8} l_i{}^2 [\hat{e}_{i+1} + (1 - a_i)]^2$ (5)	$l_i{}^2 [\hat{e}_{i+1} + (1 - a_i)]^2 S_{\hat{q}_i}{}^2$ (6)	$\sum_{j=i}^{\omega} l_j{}^2 [\hat{e}_{j+1} + (1 - a_j)]^2 S_{\hat{q}_j}{}^2$ (7)	$10^4 S_{\hat{e}_i}{}^2$ (8)
0– 1	1.4621	1	.1062	477,395	697,985	3,260,546	3.2605
1– 5	.2588	4	.4081	423,526	109,588	2,562,561	2.7053
5–10	.1717	5	.4694	363,933	62,477	2,425,973	2.8534
10–15	.2454	5	.5409	304,457	74,716	2,390,496	2.5596
15–10	.6526	5	.5316	255,140	166,511	2,315,780	2.4940
20–25	.9998	5	.5063	211,805	211,765	2,149,269	2.3446
25–30	.7873	5	.5028	169,492	133,441	1,937,504	2.1518
30–35	.8093	5	.5226	132,624	107,328	1,804,063	2.0359
35–40	1.2527	5	.5351	100,789	126,263	1,696,735	1.9489
40–45	2.0371	5	.4452	76,705	156,255	1,570,472	1.8518
45–50	3.6718	5	.5342	53,005	194,626	1,414,217	1.7389
50–55	6.5373	5	.5315	35,818	234,154	1,219,591	1.6097
55–60	10.5295	5	.5255	22,801	240,084	985,437	1.4589
60–65	17.3367	5	.5189	13,274	230,127	745,353	1.3154
65–70	28.9492	5	.5145	6,993	202,442	515,226	1.1935
70–75	47.4899	5	.5086	3,115	147,931	321,784	1.1295
75–80	88.6629	5	.4956	1,174	104,090	164,853	1.0441
80–85	172.1343	5	.4788	353	60,763	63,763	.9905

in a current life table. The data necessary for the computation are presented in table 3. These were taken from publication of the United States National Office of Vital Statistics (1955) and Bureau of the Census (1957).

Using formula (39) and referring to table 4, the essential steps in the computation are as follows:

1. Determine the sample variance of $\hat{q}_i$ (Col. 2) from

$$S_{\hat{q}_i}^2 = \hat{q}_i^2(1-\hat{q}_i)/D_i. \tag{25}$$

TABLE 5

Observed expectation of life, survival rate, and the corresponding sample standard error for white males, United States, 1955

AGE (YEARS)	OBSERVED EXPECTATION OF LIFE AT AGE x_i		x_i-YEAR SURVIVAL RATE	
x_i	$\hat{e}_i$	$S_{\hat{e}_i}$	$\hat{p}_{0_i}$	$10^3 S_{\hat{p}_{0_i}}$
0	67.3	.0181	1,00000	0
1	68.2	.0164	.97325	.11768
5	64.5	.0161	.96906	.12765
10	59.6	.0160	.96641	.13384
15	54.8	.0158	.96360	.14230
20	50.1	.0153	.95743	.16258
25	45.6	.0147	.94891	.18927
30	40.9	.0143	.94134	.20728
35	36.3	.0140	.93307	.22398
40	31.7	.0136	.92092	.24712
45	27.3	.0132	.90183	.27946
50	23.2	.0127	.87044	.32705
55	19.4	.0121	.82188	.39198
60	16.0	.0115	.75276	.46607
65	12.9	.0109	.65704	.54550
70	10.3	.0106	.53375	.62242
75	8.0	.0102	.39735	.69130
80	6.1	.0100	.25372	.74589
85	4.8	——	.12717	.75644

2. Estimate the value of a_i (Col. 4) from

$$a_i = (L_i - n_i l_{i+1})/n_i d_i. \tag{40}$$

3. Compute the term

$$l_i^2[\hat{e}_{i+1} + (1-a_i)n_i]^2 \qquad \text{(Col. 5)}.$$

4. Multiply Column 2 by Column 5 to obtain Column 6.
5. Sum these products in Column 6 from the bottom of the table up to x_i and enter the sum in Column 7.
6. Divide Column 7 by l_i^2 to obtain the sample variance of the observed expectation of life (Col. 8).

By taking the square root of the sample variance, we obtain the sample standard error of the observed expectation of life, as shown in table 5.

The x_i-year survival rate and the corersponding standard error [eq. (36)] have also been computed and are shown in the second panel of table 5.

APPENDIX

To prove the identity

$$1/l_\alpha^2\Big[\sum_{i=\alpha}^{w}(x_i+a_in_i)^2d_i-l_\alpha(\hat{e}_\alpha+x_\alpha)^2\Big]=\sum_{i=\alpha}^{w-1}\hat{p}_{\alpha i}^{\,2}[\hat{e}_{i+1}+(1-a_i)n_i]^2S_{q_i}^{\,2} \tag{47}$$

of formula (13) and (16) for the sample variance of $\hat{e}_\alpha$, we start with the left hand side of (47) and write

$$\begin{aligned}
l_\alpha^2S_{\hat{e}_\alpha}^{\,2}&=\sum_{i=\alpha}^{w}(x_i+a_in_i)^2d_i-l_\alpha(\hat{e}_\alpha+x_\alpha)^2\\
&=\sum_{i=\alpha}^{w}(x_i+a_in_i)^2(l_i-l_{i+1})-l_\alpha(\hat{e}_\alpha+x_\alpha)^2\\
&=\sum_{i=\alpha}^{w}(x_i+a_in_i)^2l_i-\sum_{i=\alpha}^{w-1}(x_i+a_in_i)^2l_{i+1}-l_\alpha(\hat{e}_\alpha+x_\alpha)^2\\
&=\sum_{i=\alpha}^{w}(x_i+a_in_i)^2l_i-\sum_{i=\alpha+1}^{w}(x_{i-1}+a_{i-1}n_{i-1})^2l_i-l_\alpha(\hat{e}_\alpha+x_\alpha)^2\\
&=\sum_{i=\alpha+1}^{w}[(x_i+a_in_i)^2-(x_{i-1}+a_{i-1}n_{i-1})^2]l_i\\
&\qquad+[(x_\alpha+a_\alpha n_\alpha)^2-(\hat{e}_\alpha+x_\alpha)^2]l_\alpha.
\end{aligned} \tag{48}$$

Since $x_{i-1}+n_{i-1}=x_i$, and $(1-a_{i-1})n_{i-1}+a_in_i=c_i$,

$$\begin{aligned}
&(x_i+a_in_i)^2-(x_{i-1}+a_{i-1}n_{i-1})^2\\
&\quad=(x_i+a_in_i-x_{i-1}-a_{i-1}n_{i-1})(x_i+a_in_i+x_{i-1}+a_{i-1}n_{i-1})\\
&\quad=c_i[2x_i-(c_i-2a_in_i)]\\
&\quad=2c_ix_i-c_i(c_i-2a_in_i),
\end{aligned}$$

and the last expression of (48) becomes

$$l_\alpha^2 S_{\hat{e}_\alpha}{}^2 = 2\sum_{i=\alpha+1}^{w} c_i x_i l_i - \sum_{i=\alpha+1}^{w} c_i(c_i - 2a_i n_i) l_i + [2a_\alpha n_\alpha x_\alpha + a_\alpha{}^2 n_\alpha{}^2 - \hat{e}_\alpha{}^2 - 2x_\alpha \hat{e}_\alpha] l_\alpha. \tag{49}$$

Now the relation

$$x_i = \sum_{h=\alpha}^{i-1} n_h + x_\alpha$$

is used and the first term in (49) is rewritten as

$$\begin{aligned} 2\sum_{i=\alpha+1}^{w} c_i x_i l_i &= 2\sum_{i=\alpha+1}^{w} c_i \Big(\sum_{h=\alpha}^{i-1} n_h\Big) l_i + 2x_\alpha \sum_{i=\alpha+1}^{w} c_i l_i \\ &= 2\sum_{h=\alpha}^{w-1} n_h \sum_{i=h+1}^{w} c_i l_i + 2x_\alpha(\hat{e}_\alpha - a_\alpha n_\alpha) l_\alpha \\ &= 2\sum_{h=\alpha}^{w-1} n_h(\hat{e}_h - a_h n_h) l_h + 2x_\alpha(\hat{e}_\alpha - a_\alpha n_\alpha) l_\alpha \\ &= 2\sum_{i=\alpha}^{w-1} n_i \hat{e}_i l_i - 2\sum_{i=\alpha}^{w-1} a_i n_i{}^2 l_i + 2x_\alpha(\hat{e}_\alpha - a_\alpha n_\alpha) l_\alpha, \end{aligned} \tag{50}$$

and (49) becomes

$$\begin{aligned} l_\alpha^2 S_{\hat{e}_\alpha}{}^2 &= 2\sum_{i=\alpha}^{w-1} n_i \hat{e}_i l_i - 2\sum_{i=\alpha}^{w-1} a_i n_i{}^2 l_i \\ &\quad - \sum_{i=\alpha+1}^{w} c_i(c_i - 2a_i n_i) l_i + (a_\alpha{}^2 n_\alpha{}^2 - \hat{e}_\alpha{}^2) l_\alpha \\ &= 2\Big[\sum_{i=\alpha}^{w-1} n_i \hat{e}_i l_i - \sum_{i=\alpha}^{w-1} a_i n_i{}^2 l_i - \sum_{i=\alpha+1}^{w} c_i(c_i - 2a_i n_i) l_i\Big] \\ &\quad + \sum_{i=\alpha+1}^{w} c_i(c_i - 2a_i n_i) l_i + (a_\alpha{}^2 n_\alpha{}^2 - \hat{e}_\alpha{}^2) l_\alpha. \end{aligned} \tag{51}$$

The quantity inside the square brackets in (51) may be rewritten as

$$\begin{aligned} &\sum_{i=\alpha}^{w-1} n_i \hat{e}_i l_i - \sum_{i=\alpha}^{w-1} a_i n_i{}^2 l_i - \sum_{i=\alpha+1}^{w} c_i(c_i - 2a_i n_i) l_i \\ &= \sum_{i=\alpha+1}^{w} n_{i-1} \hat{e}_{i-1} l_{i-1} - \sum_{i=\alpha+1}^{w} a_{i-1} n_{i-1}{}^2 l_{i-1} \\ &\quad - \sum_{i=\alpha+1}^{w} [(1 - a_{i-1})^2 n_{i-1}{}^2 - a_i{}^2 n_i{}^2] l_i \\ &= \sum_{i=\alpha+1}^{w} (n_{i-1} - a_{i-1} n_{i-1}) [\hat{e}_{i-1} l_{i-1} - a_{i-1} n_{i-1} l_{i-1} - (1 - a_{i-1}) n_{i-1} l_i] \\ &\quad + \sum_{i=\alpha+1}^{w} a_i n_i \hat{e}_i l_i + (a_\alpha n_\alpha \hat{e}_\alpha - a_\alpha{}^2 n_\alpha{}^2) l_\alpha \end{aligned} \tag{52}$$

$$= \sum_{i=\alpha+1}^{w} (n_{i-1} - a_{i-1}n_{i-1})\hat{e}_i l_i + \sum_{i=\alpha+1}^{w} a_i n_i \hat{e}_i l_i + (a_\alpha n_\alpha \hat{e}_\alpha - a_\alpha^2 n_\alpha^2) l_\alpha$$

$$= \sum_{i=\alpha+1}^{w} [(1 - a_{i-1})n_{i-1} + a_i n_i]\hat{e}_i l_i + (a_\alpha n_\alpha \hat{e}_\alpha - a_\alpha^2 n_\alpha^2) l_\alpha$$

$$= \sum_{i=\alpha+1}^{w} c_i \hat{e}_i l_i + (a_\alpha n_\alpha \hat{e}_\alpha - a_\alpha^2 n_\alpha^2) l_\alpha,$$

where the relationship $[\hat{e}_{i-1} - a_{i-1}n_{i-1}]l_{i-1} = [\hat{e}_i + (1 - a_{i-1})n_{i-1}]l_i$ has been used. Substituting (52) in (51) gives

$$l_\alpha^2 S_{\hat{e}_\alpha}^2 = 2\Big[\sum_{i=\alpha+1}^{w} c_i \hat{e}_i l_i + (a_\alpha n_\alpha \hat{e}_\alpha - a_\alpha^2 n_\alpha^2) l_\alpha\Big] + \sum_{i=\alpha+1}^{w} c_i(c_i - 2a_i n_i) l_i + (a_\alpha^2 n_\alpha^2 - \hat{e}_\alpha^2) l_\alpha \qquad (53)$$

$$= \sum_{i=\alpha+1}^{w} [2c_i \hat{e}_i + c_i(c_i - 2a_i n_i)] l_i - (\hat{e}_\alpha - a_\alpha n_\alpha)^2 l_\alpha,$$

where

$$\begin{aligned} 2c_i\hat{e}_i + c_i(c_i - 2a_i n_i) &= 2\{(1 - a_{i-1})n_{i-1} + a_i n_i\}\hat{e}_i + \{(1 - a_{i-1})^2 n_{i-1}^2 - a_i^2 n_i^2\} \\ &= \{\hat{e}_i + (1 - a_{i-1})n_{i-1}\}^2 - \{\hat{e}_i - a_i n_i\}^2 \\ &= \{\hat{e}_i + (1 - a_{i-1})n_{i-1}\}^2 - \{\hat{e}_{i+1} + (1 - a_i)n_i\}^2 \hat{p}_i^2 \end{aligned}$$

and $\hat{e}_\alpha - a_\alpha n_\alpha = \{\hat{e}_{\alpha+1} + (1 - a_\alpha)n_\alpha\}\hat{p}_\alpha$. Consequently,

$$\begin{aligned} l_\alpha^2 S_{\hat{e}_\alpha}^2 &= \sum_{i=\alpha+1}^{w} \{\hat{e}_i + (1 - a_{i-1})n_{i-1}\}^2 l_i - \sum_{i=\alpha}^{w} \{\hat{e}_{i+1} + (1 - a_i)n_i\}^2 \hat{p}_i^2 l_i \\ &= \sum_{i=\alpha}^{w-1} [\{\hat{e}_{i+1} + (1 - a_i)n_i\}^2 l_{i+1} - \{\hat{e}_{i+1} + (1 - a_i)n_i\}^2 \hat{p}_i^2 l_i] \qquad (54) \\ &= \sum_{i=\alpha}^{w-1} \{\hat{e}_{i+1} + (1 - a_i)n_i\}^2 (l_{i+1} - \hat{p}_i^2 l_i) \\ &= \sum_{i=\alpha}^{w-1} \{\hat{e}_{i+1} + (1 - a_i)n_i\}^2 l_i^2 S_{\hat{q}_i}^2, \end{aligned}$$

where

$$S_{\hat{q}_i}^2 = (1/l_i)\hat{p}_i(1 - \hat{p}_i).$$

The last expression in (54) divided by l_α^2 is identical with the right hand side member of identity (47) and the proof is complete.

LITERATURE CITED

BUREAU OF THE CENSUS 1957 Estimates of the population of the United States, by age, color, and sex. July, 1955 to 1957. Current Population Reports, Series P-25, No. 170.

CHIANG, C. L. 1958 An application of stochastic processes to the life table and standard error of age adjusted rate (abstract), Biometrics, *14*: 133-134.

——— In press A A stochastic study of the life table and its applications. I. Probability distributions of the biometric functions. Biometrics.

——— In press B Standard error of the age adjusted death rate (to be published as a special report, National Office of Vital Statistics).

——— In press C A stochastic study of the life table and its applications. III. The follow-up study with the consideration of competing risks. Biometrics.

DOERING, C. R., AND A. L. FORBES 1938 A skeleton life table. Proc. National Acad. Sci., *24*: 400-405.

ELVEBACK, L. 1958 Estimation of survivorship in chronic disease: the "actuarial" method. J. Amer. Statistical Assoc., *53*: 420-440.

GREENWOOD, M. 1926 A report on the natural duration of cancer. Reports on Public Health and Medical Subjects, No. 33, His Majesty's Stationery Office, pp. 1-26.

GREVILLE, T. N. E. 1943 Short methods of constructing abridged life table. Record of the American Institute of Actuaries, *32*: 29-42.

IRWIN, J. O. 1949 The standard error of an estimate of expectational life. J. Hygiene, *47*: 188-189.

KAPLAN, E. L., AND P. MEIER 1958 Nonparametric estimation from incomplete observations. J. Amer. Statistical Assoc., *53*: 457-481.

LITTELL, A. S. 1952 Estimation of the *t*-year survival rate from follow-up studies over a limited period of time. Human Biol., *24*: 87-116.

MILLER, R. S., AND J. L. THOMAS 1958 The effects of larval crowding and body size on the longevity of adult *Drosophila melanogaster*. Ecology, *39*: 118-125.

NATIONAL OFFICE OF VITAL STATISTICS 1955 Vital Statistics of the United States, vols. I and II.

REED, L. J., AND M. MERRELL 1939 A short method for constructing an abridged life table. Amer. J. Hygiene, *30*: 33-62.

WILSON, E. B. 1938 The standard deviation of sampling for life expectancy. J. Amer. Statistical Assoc., *33*: 705-708.

SUBCUTANEOUS TISSUE IN POLYNESIAN CHILDREN FROM RAROTONGA, COOK ISLANDS

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INTRODUCTION

MEASUREMENT of the amount and distribution of subcutaneous tissue is now a routine part of many studies of human growth (Keys, 1956; Garn and Shamir, 1958). Published reports on subcutaneous tissue in children deal mainly with subjects from the Western world, however, and information on other populations is scarce.

Data are presented in this article on the amount and distribution of subcutaneous tissue in 221 Polynesian boys and girls from the island of Rarotonga, Cook Islands, South Pacific. Measurements of 129 Rarotongan boys were made at the waist, chest, back, forearm and upper arm. Measurements of 92 Rarotongan girls were made, because of their modesty, only at the upper arm.

Comparative material

Articles containing reviews of the literature on subcutaneous tissue have been presented by Brožek and Keys (1950/1951) and Reynolds (1951). Both articles include extensive bibliographies. For this report, comparative material on the amount and distribution of subcutaneous tissues has been taken from studies of children in Iowa (Meredith, 1935; Boynton, 1936), Ohio (Reynolds, 1951) and England (Hammond, 1955).

METHODS

Subjects

A total of 129 Rarotongan boys and 92 Rarotongan girls between 5 and 20 years of age were measured. It has been reported that 59% of the Cook Islanders are mixed in some way with non-Polynesian racial elements (Simmons, Graydon, Semple and Fry, 1955). Morphologically, the 221 Polynesian children included in this study reflect this foreign admixture, and are thus representative of the Cook Island population as a whole.

Skin fold measurement techniques

All measurements were made with a figure-eight spring-operated caliper of the type used by Brožek and Keys (1950/1951), Newman (1952), and Baker (1955). In using these calipers, the observer picks up a "bite" of skin and subcutaneous tissue about 4 cm wide between his left thumb and forefinger. The calipers are applied with the right hand and allowed to grip the fold. The left hand is then removed, and the fold is held with the calipers while the reading is taken. Three determinations are made at each site, each time an attempt being made to pick up the same amount of tissue at the same location. The three observations were averaged, and the mean value used. All measurements mere made on the right side, with the subject standing.

Upper arm fold: Taken at the back of the upper arm a few inches below the axillary fold, along the long axis of the arm.

Forearm fold: Taken at the most lateral edge of the upper portion of the forearm in the anatomical position, along the long axis of the arm.

Chest fold: Taken midway along a diagonal line connecting the axilla and nipple.

Waist fold: Taken along the waist line, halfway between the umbilicus and the most lateral part of the waist.

Back fold: Taken on the back just below the inferior border of the scapula, parallel to the vertebral column.

RESULTS

Table 1 presents two mean values for each body area measured. The first value represents the actual caliper reading; the second is the x-ray transformation of the caliper reading using Baker's (1955) formula.

The small number of subjects in each age group results in large fluctuations of the mean values from year to year. In figure 1, three-point moving averages were used to smooth the fluctuations and to reveal the general trend more clearly.

These data show that subcutaneous tissue in Rarotongans increases with advancing age. The general trend of these increases is accelerated during adolescence, but slows down during the last year of measurement. Decreases are seen only in the measurement of the upper arm of the girls from 6 to 9 years and from 18 to 19 years. This latter loss is probably an artifact due to the small number of cases measured.

TABLE 1

Mean caliper and X-ray values of Rarotongan subcutaneous tissue (mm)

TABLE 1

Mean caliper and X-ray values of Rarotongan subcutaneous tissue (mm)

AGE	NO.	UPPER ARM		FOREARM		CHEST		WAIST		BACK	
		CALIPER	X-RAY	CALIPER	X-RAY	CALIPER	X-RAY	CALIPER	X-RAY	CALIPER	X-RAY
BOYS											
6	8	7.54	6.34	6.46	5.68	4.46	4.45	4.38	4.40	5.12	4.85
7	12	6.53	5.72	6.03	5.41	4.58	4.52	4.86	4.69	5.57	5.13
8	6	6.89	5.94	6.00	5.39	4.94	4.74	5.50	5.09	6.00	5.39
9	8	7.78	6.49	6.67	5.80	4.50	4.47	6.08	5.44	5.54	5.11
10	8	7.67	6.42	6.46	5.68	6.21	5.52	6.25	5.55	7.42	6.26
11	5	7.40	6.25	6.87	5.93	5.55	5.12	5.53	5.10	8.33	6.82
12	6	8.89	7.17	7.17	6.11	6.44	5.66	7.79	6.49	9.22	7.37
13	11	8.42	6.88	7.48	7.05	6.30	5.58	7.39	6.25	8.09	6.68
14	12	9.06	7.27	7.86	6.54	6.47	5.68	7.39	6.25	9.55	7.57
15	13	9.81	7.73	8.44	6.89	7.49	6.31	9.90	7.79	11.23	8.60
16	13	10.18	7.96	8.69	7.04	7.46	6.29	9.82	7.74	11.15	8.56
17	6	12.94	9.66	10.00	7.85	9.61	7.61	12.61	9.45	13.89	10.24
18	6	14.39	10.54	10.44	8.02	9.44	7.51	12.33	9.28	14.94	10.88
19	8	15.25	11.07	10.67	8.26	11.13	8.54	14.62	10.69	18.33	12.96
20	7	15.29	11.10	9.14	7.32	9.81	7.73	13.14	9.78	17.38	12.38
GIRLS											
5	2	9.34	7.44	no data		no data		no data		no data	
6	12	9.17	7.34								
7	5	9.67	7.65								
8	5	9.33	7.45								
9	11	9.00	7.24								
10	10	9.75	7.70								
11	6	12.56	9.42								
12	7	10.29	8.03								
13	8	11.63	8.85								
14	8	12.83	9.59								
15	4	14.67	10.72								
16	2	14.84	10.82								
17	4	18.58	13.12								
18	1	19.33	13.58								
19	3	25.11	17.13								
20	4	21.75	15.06								

It is difficult to compare the Rarotongan results with those from other groups; some studies used x-rays, while in others different types of calipers were used. Moreover, the sites measured rarely agree. The

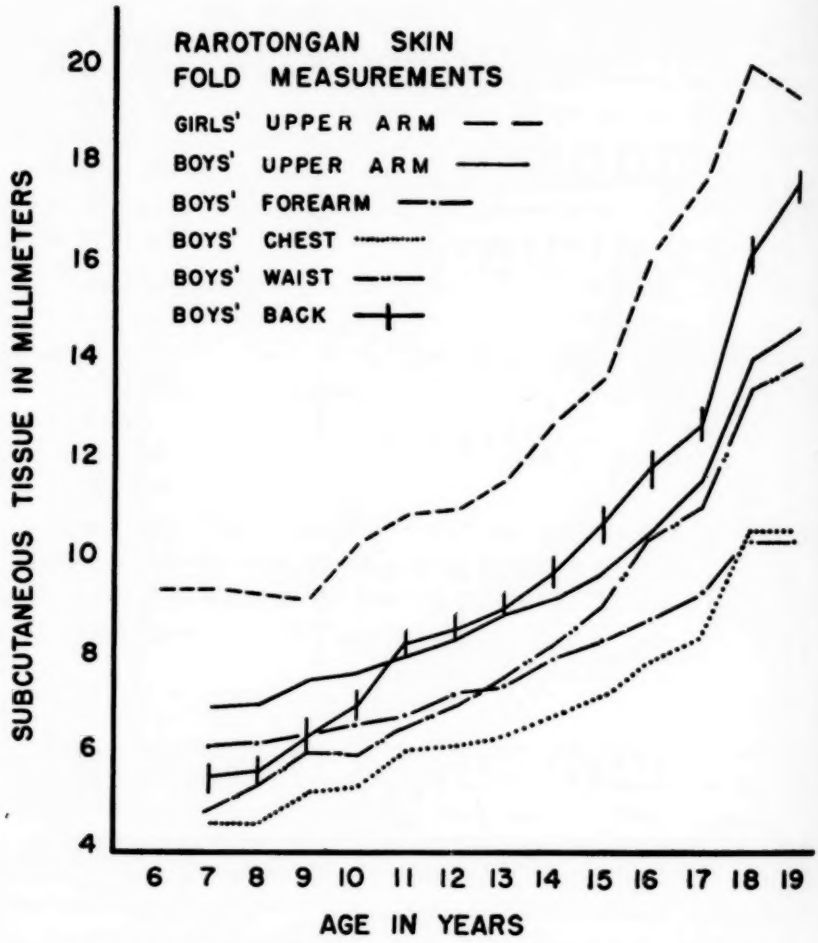

FIG. 1. MOVING AVERAGE AGE-MEANS OF RAROTONGAN SKIN FOLDS.

upper arm measurement is the only one which is located in even approximately the same area in the comparative studies presented here. Because of this lack of comparability, a number of reports could not be used (Stuart and Sobel, 1946; Stolz and Stolz, 1951; Garn and Haskell, 1959).

TABLE 2

Upper arm subcutaneous tissue (mm) in Iowan, English, Ohio and Rarotongan children

Boys					Girls				
AGE	IOWA [1]	ENGLAND [2]	OHIO [3]	RAROTONGA [3]	AGE	IOWA [1]	ENGLAND [2]	OHIO [3]	RAROTONGA [3]
7	6.1	7.40	5.15	5.72	7	6.97	8.62	6.35	7.65
8	6.2	7.11	5.30	5.94	8	7.45	9.23	6.55	7.45
9	6.4	7.67	5.55	6.49	9	7.88	9.35	6.85	7.24
10	6.4	7.66	5.75	6.42	10	7.82	9.44	7.20	7.70
11	6.9	8.26	6.15	6.25	11	7.70	9.64	7.30	9.42
12	6.9	7.87	6.35	7.17	12	7.71	9.21	7.25	8.03
13	6.7	7.59	6.15	6.88	13	7.36	9.45	7.45	8.85
14	6.4	7.42	5.70	7.27	14	7.99	10.13	7.80	9.59
15	5.8	5.97	5.25	7.73	15	8.65	10.19	8.65	10.72
16	5.4	5.90	5.25	7.96					
17	5.5	6.06	5.60	9.66					

[1] Calipers.

[2] Calipers converted to x-ray.

[3] X-ray. Interpolation from measurements reported at the mid-year.

Table 2 presents a comparison of upper arm subcutaneous tissue as measured by x-ray or calipers on children from Iowa, Ohio, England and Rarotonga. The table lists only those ages which were common to all studies.

Subcutaneous tissue deposit in the upper arm of the Rarotongan boys appear to be larger than in most of the comparative groups, but more importantly, the Rarotongan pattern of tissue gains is very different. All of the groups of boys show small gains in pre-adolescence, but only the Rarotongans continue substantial increases into adolescence. The other groups of boys show an adolescent decrease in the thickness of subcutaneous tissue, yet the Rarotongans make their biggest gains during this time period.

The Rarotongan girls agree with the other groups in showing steady increases in pre-adolescence, and a large adolescent increase. In general, Rarotongans of both sexes appear to be more similar to the girls of the comparative groups than to the boys.

The allometry equation, $y = bx^k$ (Richards and Kavanagh, 1945) was applied to the Rarotongan data in order to determine the age at which the adolescent spurt in subcutaneous tissue growth occurs. The equation was used in its logarithmic form, $\log y = \log b + k \ (\log x)$, and calculated by the method of least squares (Zucker, 1947). In these analyses, the skin fold data were compared both to body weight and to chronological age. The k values resulting from the calculation of the equation show the slope of the line best describing the relationship between the two dimensions under consideration. When the k value exceeds 1.00, the ordinate dimension is increasing faster than the dimension on the abcissa, and the converse is true when the k value is less than 1.00. In all cases, skin folds are the ordinate dimension, and either weight or age the abcissa dimension. The Rarotongan k values are shown in table 3.

In every case except log chest fold vs. log weight, the slope of the allometric line increased indicating that a continuing process of acceleration occurred. Prior to the growth spurt during adolescence, the velocity of weight increases exceeded that of subcutaneous tissue increases. In this time interval, all k values were less than 1.00. During adolescence, the velocity of gains in subcutaneous tissue exceeded that of gains in weight, and k was larger than 1.00. This general statement does not hold true for the chest fold of the boys or the upper arm fold of the girls. The chest fold of boys shows a decreased slope during adolescence. In pre-adolescence, the arm fold of girls shows a slight negative slope, as weight is gained, but subcutaneous tissue is lost.

In general, large gains in weight are not made after 16 years of age. This is the time period, however, when the amount of subcutaneous tissue shows a sudden spurt. It is evident, therefore, that most weight gains after 16 years of age result in the deposition of subcutaneous tissue. This process of tissue deposition is shared by all of the body areas measured, with the sub-scapular area receiving the most, and the forearm area the least.

The onset and end of the adolescent growth spurt are indicated by changed slopes of the allometric line describing the relationship between the dimensions. The ages at which these changes of slope appear and disappear are given in table 4.

The dimensions with an age listed under "Age at End" are those with a definite spurt appearing during adolescence. It will be seen that five of the six skin fold vs. age measurements had such a spurt, while only two of the six skin fold vs. weight measurements had such a spurt. This would indicate that increases in subcutaneous tissue tend to be timed somewhat independently of gains in body weight and confirms similar results found by Hammond (1955) on English children.

When skin folds are compared to age, the adolescent spurt began, on the average, at 12.5 years for the boys. For the girls, 9.5 years is the time of onset for the one available dimension. The average age at the end of the adolescent spurt is 16.25 years for the boys, and 13.5 years for the girls.

There is no general spurt in adolescence when the skin fold dimensions vs. weight are considered. Only boys' chest fold and girls' upper arm fold show such a spurt.

SUMMARY

Caliper measurements of subcutaneous tissue were made on five body areas of 129 Polynesian boys 5 to 20 years of age from the island of Rarotonga, Cook Islands, South Pacific. One area was measured on 92 Rarotongan girls. The Rarotongan boys show a pattern of continuous tissue gains in childhood and adolescence, whereas most comparative groups make only slight gains during these periods. From 6 to 9 years, the Rarotongan girls lose subcutaneous tissue from their upper arm, but gain thereafter. The girls' pattern of gains is generally similar to that of other groups.

An adolescent spurt of growth was seen for five or six dimensions when the allometric equation was applied to subcutaneous tissue and age. The average age of the Rarotongan boys at the onset of this spurt

TABLE 3

Rarotongan skin fold k values

DIMENSION	SKIN FOLD VS. WEIGHT AGE	SKIN FOLD VS. WEIGHT *k*	SKIN FOLD VS. AGE AGE	SKIN FOLD VS. AGE *k*
Boys				
Waist	7-15	0.74	7-10	0.64
	16-19	1.79	11-16	1.22
			15-19	1.86
Chest	7-12	0.68	7-16	0.65
	12-15	0.43	15-19	1.74
	15-19	1.46		
Back	7-17	0.85	7-13	0.88
	17-19	2.69	12-18	1.48
			17-19	2.93
Forearm	7-16	0.41	7-12	0.30
	16-19	1.04	11-17	0.70
			16-19	1.08
Upper Arm	7-16	0.45	7-13	0.40
	16-19	1.91	12-15	0.71
			16-19	2.11
Girls				
Upper Arm	6-9	—0.08	6-9	—0.55
	9-15	0.58	10-14	0.59
	15-19	2.01	13-19	1.51

TABLE 4

Age changes of Rarotongan skin fold scores

DIMENSION	SKIN FOLD VS. WEIGHT Age at Onset	SKIN FOLD VS. WEIGHT Age at End	SKIN FOLD VS. AGE Age at Onset	SKIN FOLD VS. AGE Age at End
Boys				
Waist	15.5	—	10.5	15.5
Chest	12.0	15.0	15.5	—
Back	17.0	—	12.5	17.5
Forearm	16.0	—	11.5	16.5
Upper Arm	16.0	—	12.5	15.5
Girls				
Upper Arm	9.0	15.0	9.5	13.5

was 12.5 years. Growth spurts were not common when subcutaneous tissue was compared to weight.

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LITERATURE CITED

BAKER, P. T. 1955 Relationship of Desert Heat Stress to Gross Morphology. Quartermaster Res. & Develop. Center, Natick, Mass., Technical Rep. EP-7.

BOYNTON, B. 1936 The Physical Growth of Girls. Univ. of Iowa Studies in Child Welfare, vol. 12, number 4.

BROŽEK, J., AND A. KEYES 1950/1951 Evaluation of leanness-fatness in man: a survey of methods. Nutrition Abstr. Rev., *20*: 247-256.

——— 1951 The evaluation of leanness-fatness in man: norms and interrelationships. Brit. J. Nutrition, *5*: 194-206.

GARN, S. M., AND J. A. HASKELL 1959 Fat changes during adolescence. Science, *129*: 1615-1616.

GARN, S. M., AND Z. SHAMIR 1958 Methods for Research in Human Growth. Charles C. Thomas, Springfield, Illinois.

HAMMOND, W. H. 1955 Measurement and interpretation of subcutaneous fat with norms for children and young adult males. Brit. J. Preventive and Social Med., *9*: 201-211.

KEYS, A. 1956 Recommendations concerning body measurements for the characterization of nutritional status. Human Biol., *28*: 111-123.

MEREDITH, H. V. 1935 The rhythm of physical growth. Univ. of Iowa Studies in Child Welfare, vol. 11, number 3.

NEWMAN, R. W. 1952 Measurement of Body Fat in Stress Situations. Office of the Quartermaster General, Res. & Develop. Division, Environmental Protection Branch, Report No. 193.

REYNOLDS, E. L. 1951 The Distribution of Subcutaneous Fat in Childhood and Adolescence. Monographs of the Society for Research in Child Development, vol. 15, number 2.

RICHARDS, O. W., AND A. J. KAVANAGH 1945 The analysis of growing form. Pp. 188-230 of Essays on Growth and Form, Essays Presented to D'Arcy Wentworth Thompson. W. E. LeGros Clark and P. B. Medawar (Eds.). Clarendon Press, Oxford.

SIMMONS, R. T., J. J. GRAYDON, N. M. SEMPLE AND E. I. FRY 1955 A blood

group genetical survey in Cook Islanders, Polynesia, and comparisons with American Indians. Am. J. Phys. Anthrop., n. s. *13*: 743-746.

STOLZ, H. R., AND L. M. STOLTZ 1951 Somatic Development of Adolescent Boys. The Macmillan Co., New York.

STUART, H. C., AND E. H. SOBEL 1946 The thickness of the skin and subcutaneous tissue by age and sex in childhood. J. Pediatrics, *28*: 637-647.

TANNER, J. M., AND R. H. WHITEHOUSE 1955 The Harpenden skinfold caliper. Am. J. Phys. Anthrop., n. s. *13*: 743-746.

ZUCKER, L. M. 1947 Evaluation of slopes and intercepts of straight lines. Human Biol., *19*: 231-259.

THE TABOR HILL OSSUARIES: A STUDY IN IROQUOIS DEMOGRAPHY

BY C. S. CHURCHER[1] AND W. A. KENYON[2]

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The Archaeological and Historical Sites Board of Ontario, which placed the site under its authority, was largely responsible for protecting this find from unauthorized digging and vandalism. The Royal Ontario Museum and the Department of Zoology, University of Toronto, were most helpful in providing the various facilities that we have enjoyed while working on the project.

INTRODUCTION

Tabor Hill, situated on the eastern watershed of Highland Creek, Scarborough Township, within Metropolitan Toronto, is the highest feature in the area (figure 1). The elevation of the hill as indicated on the topographic maps of the region is between 550 and 575 feet above sea-level; its major axis lies approximately 125-305° Grid North. The site lies approximately one quarter of the way from the southeastern end of the hill at an elevation of some 560 feet. Alternatively, the site is located between Woburn and Bendale on Concession 1, Lot 20, Scarborough Township.

Description of Site.

The site was discovered in September, 1956, during earth-moving operations connected with the residential development of the section of land on the northwest corner of the intersection of Bellamy Road and

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[2] Royal Ontario Museum, Toronto 5, Canada.

Lawrence Avenue East, when a power shovel accidently uncovered some human bones. The Royal Ontario Museum was informed and asked to investigate. On examination, the bones proved to have come from two separate ossuaries whose methods of burial left no doubt as to their being Iroquois in origin. Some of these bones were still in situ within the

FIGURE 1. MAPS SHOWING THE LOCATION OF THE TABOR HILL OSSUARIES IN ONTARIO AND IN RELATION TO THE MAIN ROADS OF THE AREA.

ossuaries, but many had fallen to the foot of the earthface formed by the power shovel's excavations. Many whole and broken bones lay around the site; others had been collected by the local police for safe keeping; and others had been pilfered by souvenir hunters.

Because of the approach of winter and because of the position of the site within a housing development, the archaeological operation was in

the nature of a rough and ready salvage of all obtainable material. This was accomplished over a period of a few days and consequently all the care and precautions normally taken in an excavation of this sort could not be applied. In addition, because of the 'treasure-hunting' by the local population the sample obtained is probably biased, or at least suffers from inadequacies, and this should be borne in mind throughout this report.

All the loose bones were collected and added to all those collected by the police and those retrieved from the local populace. The ossuaries were cleaned by removal of the disturbed soil and the faces of both ossuaries profiled. Sketches of the faces were made, and measurements of the cross-sectional proportions obtained. The extent of both ossuaries was then determined by the expedient of digging narrow trenches towards the exposed bones from a number of directions. The shape and size of both ossuaries was thus determined fairly early in the operation.

The larger ossuary was oval in plan (figure 2), with major and minor diameters of approximately 13.5 feet and 10 feet. Only about one half of this pit remained when salvage began. The pit was flat-bottomed in section, about 3.6 feet deep, with rounded corners and vertical sides. The stratum of bone in the bottom of the pit was roughly 2 feet deep and was overlain by a layer of back-filled soil about 1.6 feet deep.

The smaller ossuary was a lobate structure (figure 2), with a maximum dimension of some 9 feet; its greater lobe was about 6 feet in diameter, its lesser lobe about 4 feet. An estimated fifth of this ossuary was missing. The section revealed by the power-shovel showed that the pit had a rather uneven bottom, sloping walls, and a depth of some 2.25 feet. This pit was clearly a less perfect structure than the larger ossuary. The two pits were 38 feet apart, centre to centre, separated by 28.5 feet of undisturbed soil, and with their long axes lying nearly along the ridge of Tabor Hill. Because the overburden, humus and other organic matter, had been stripped from the hill by the contractors prior to the discovery of the ossuaries, their original depth can only be estimated.

The soil in which the ossuaries were situated is greyish-brown in colour, and of glacial derivation. It has been identified as a slightly acid sandy loam, containing few stones, and derived from a medium textured shaly calcareous till. It belongs to the grey-brown podzolic grouping of soils which are common in southern Ontario (Hoffman and Richards, 1955). The topography of the district is of glacial origin and contains numerous poorly developed drumlins, Tabor Hill itself being a low drumlin.

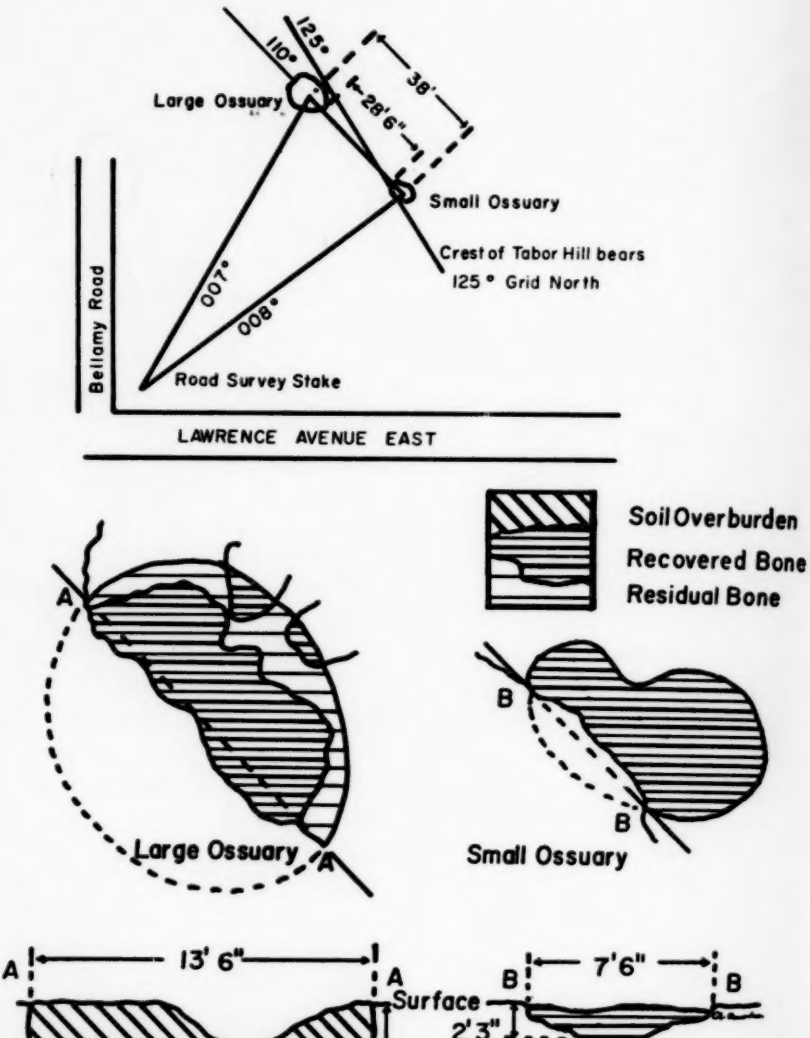

FIGURE 2. PLAN LOCATING THE TWO TABOR HILL OSSUARIES RELATIVE TO THE INTERSECTION OF BELLAMY ROAD AND LAWRENCE AVENUE EAST. SKETCH MAPS AND SECTIONS OF THE TWO OSSUARIES TO SHOW DEPTH OF BURIAL, PROBABLE EXTENT AND PROPORTION OF BONES SALVAGED.

Heavy earthmoving equipment had been used to remove the top soil and had removed an estimated eight inches from the surface according to one caterpillar operator. While this estimate may not be considered completely reliable, it is the only measure available since no remnants of top-soil remained in the near area. The addition of eight inches of soil above the observed surface would mean that the larger ossuary had been covered originally by about 2.3 feet of soil and the smaller by only eight inches of soil. This latter figure seems too small, and the explanation may lie in additional loss of top-soil during the agricultural tilling of the field by European settlers during the last 150 years. Because of the greater depth of soil over the larger ossuary, the frequency of recent breakage was lower in this ossuary than in the smaller, although considering the weight of the machines used in earth-moving the frequency in either ossuary was very low. This difference in breakages was noted by both authors during the abstraction of the bones from both ossuaries. The burial customs of the Iroquois are too well known to warrant description here; the interested reader will find an excellent summary statement in Kidd (1953). A few comments on the distribution of the bones within the ossuaries, however, might be profitable. As a rule, the bones were randomly scattered throughout the pits, with no attempt to keep the remains of any single individual together. Possibly the skeletons were carried to the edge of the pits in baskets or similar containers, and then strewn over the bottom of the grave. Several bundle-burials were present, however, suggesting that at least the long-bones of some individuals had been thrown into the pit while tied into a neat package. In two instances, pockets of burned bone attested to the presence of cremations.

Shortly after the Tabor Hill ossuaries were discovered, an Iroquois village was found about a mile and a quarter upstream, on the north shore of Highland Creek. It is situated on the west side of Brimley Road, on the William Thompson farm. A brief but fairly comprehensive archaeological survey of the region suggests that this is the only large village-site within a radius of several miles. There is a very high probability, therefore, that this was the main village of the people whose bones we recovered from Tabor Hill.

Over the week-end of October 20th, Dr. J. Norman Emerson, Department of Anthropology, University of Toronto, partially excavated this site. On the basis of pottery types present, as well as a wide range of other artifacts, he was able to suggest an approximate date for this site of about 1250 A. D.

Material.

The skeletal material recovered from the two ossuaries is fairly light in weight, and stained a golden-brown, as is normal for bones taken from graves and ossuaries in the sandy loams of southern Ontario. These bones were generally well preserved and quite strong, little damage being present dating from the original interment and the bones having suffered only slight decalcification. Recent fractures were plentiful, mainly on the longbones, ribs and crania in the smaller ossuary, which could be attributed to the passage of heavy equipment over them, as mentioned earlier. Evidence of a number of diseases of the bone itself, the joints and teeth were observed, but no clear evidences of violence to the skeleton when alive. It should also be mentioned that no evidences of working were found on any of the bones, nor were any pottery, bone, stone, or metal artifacts or trade goods recovered from either of the ossuaries.

This paper will be concerned with the normal attributes of the skeletons only, all pathological or abnormal specimens or conditions being ignored for the present, and being held back for further possible work.

Although elements of all parts of the skeleton were present in the sample obtained, with the exception of hyoid bones, measurements are limited to the whole and fragmentary specimens tabulated here (table 1).

TABLE 1

Census of Whole and Fragmentary Bones from the Tabor Hill Ossuaries.

BONE	NUMBER
Crania	90
Humeri	403
Radii	196
Ulnae	240
Femora	592
Tibiae	365
Astragali (Tali)	97
Calcanei	79

These bones originated from both ossuaries, but because of the conditions under which the material was obtained, the materials salvaged from each ossuary could not be kept separate. Throughout this paper, therefore, the materials from both ossuaries will be treated as a single unit.

In general, throughout this paper, the methods, dimensions and pro-

cedures given in Hrdlička (1952) and Krogman (1946) have been followed whenever assigning age or sex to specimens.

Sexing was based on the following criteria: size and general massiveness, prominence of muscular attachments, and development of the supra-orbital tori. The only internal check on the reliability of our sexing was provided by the relatively large sample of femora and femur heads. The 61 complete femora were first sexed on the basis of the subjective criteria mentioned above. Then, after the sex of each specimen was recorded, the antero-posterior diameter of the heads was measured, producing the bi-modal distribution shown in Figure 3. Finally, the same

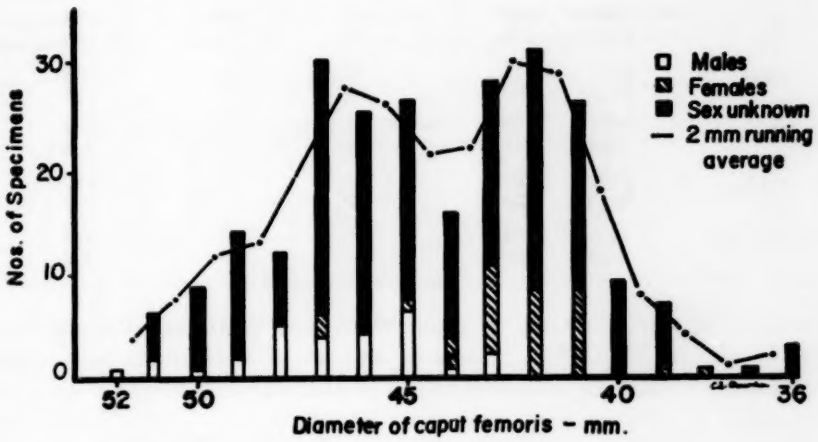

FIGURE 3. HISTOGRAM OF THE DIAMETERS OF THE CAPUT FEMORIS OF FEMORA OF KNOWN AND UNKNOWN SEX. A 2 MM RUNNING AVERAGE HAS BEEN DRAWN TO EMPHASIZE THE BIMODAL NATURE OF THE DISTRIBUTION.

measurement was made on the 184 proximal fragments of unknown sex, producing the same bi-modal distribution. This agreement suggests that the sexes assigned to the specimens are relatively reliable, and that any mistakes in sexing are the results of a common error present in both methods.

The word 'maturity,' as generally used throughout this paper, refers to bones and parts of bones, not to individuals. Age estimated for the crania and cranial fragments are based on suture closure and pattern of tooth eruption: for the longbones, epiphysial union was used.

Number of individuals.

The determination of the minimum number of individuals from which this sample could have been obtained was based upon a count of the proximal and distal fragments and the whole longbones obtained (table 2). The reliance upon longbones was founded upon their relative abund-

TABLE 2

Census of Long Bones from Tabor Hill Ossuaries to Determine the Minimum Number of Individuals from which this Sample could have been Obtained.

BONE		COMPLETE	FRAGMENTARY PROXIMAL	FRAGMENTARY DISTAL	MAXIMUM TOTALS
Radius	Left	20	44	42	64
	Right	20	35	35	55
Ulna	Left	17	81	13	98
	Right	23	93	13	116
Humerus	Left	40	61	103	101
	Right	40	64	95	104
Tibia	Left	26	91	70	117
	Right	34	83	61	117
Femur	Left	48	165	103	213
	Right	55	124	98	179

ance, their excellent state of preservation, and their ease of recognition as to side of body. The minimum number of individuals from which this sample could have been obtained is 213, which is founded upon the presence of 165 proximal fragments of left femora and 48 whole left femora.

Table 3 gives the distribution of fragmentary end entire crania by sex and age. The age range is about 50 years, and the sample consists of 38 non-adult and 52 adult specimens, or 90 specimens in all. Knowles (1937) states that Indian children develop more rapidly than do children of white parents, and therefore it is probable that the ages assigned in table 3 underestimate the actual ages of the specimens at death. On the basis of cranial identification, the proportion of immature individuals recovered from the Tabor Hill Ossuaries, with an upper limit of 16 years, is 10%. On the basis of the postcranial skeleton, the proportion of immature individuals is 29.8%. This difference may reflect the easier destruction of infant crania by weathering and decay, as the cranial bones are very soft.

It is evident in table 3 that there are two periods during which death

seems more likely to occur than during the remainder of the individual's life. The first is between the ages of 16 and 25 years, and is mainly restricted to the female group. This might be explained as a result of death during childbirth. The absence of any visible increase in the deathrate of young males between these ages might be explained in terms of their probable habits. Death for a young warrior might occur in battle or in a hostile village after being taken prisoner. If he belonged to the winning side, his body might either have been taken back to the village for burial or buried on the battleground. If his body became the property of the enemy, then his bones might have been consigned to the refuse

TABLE 3

Distribution of the Population by Age and Sex, Based on the Cranial Material Recovered. Note Small Number of Males Recovered from the 16-25 year groups.

AGE IN YEARS	SEX MALE	FEMALE	?	TOTAL NO.
Infant			5	5
10–15			4	4
16–20	6	21	2	29
21–25	4	12		16
26–30		2		2
31–35	1	1		2
36–40	3	2		5
41–45	6	4	1	11
46–50	1	3		4
50–inf				
Unknown age	6	5	1	12
Totals	27	50	13	90

dump of the hostile village or left to rot in the forest. In any of these cases there would have been a reduction in the proportion of the male skeletons within the common ossuary, and one would not expect to find an equivalent number of male and female crania in the ossuaries for these age groups. However, if it is assumed that approximately equal numbers of males and females were born and survived to puberty, it can be postulated that because there were 33 females and only 10 males recovered from the ossuaries for the age group 16-25 years, a further 23 males should be added to the count to give a true reflection of the composition of the population. This would increase the total number of individuals represented by the crania recovered from the ossuaries from 90 to 113.

The second period during which death seems more likely to occur is from the ages of 36 to 50 years, when the numbers of male to female deaths are about equal. Death at this age is probably attributable to old age, and thus an equal male to female ratio is expected and is obtained, *i. e.*, 10 males to 9 females.

The postcranial material can be broken down further into smaller groups (table 4). The minimum number of individuals required to give

TABLE 4

Census of Long Bones from the Tabor Hill Ossuaries Broken Down to Show the Distribution of Immature and Mature Skeletal Elements.

BONE		ENTIRE		FRAGMENTARY PROXIMAL		FRAGMENTARY DISTAL	
		IMMATURE	ADULT	IMMATURE	ADULT	IMMATURE	ADULT
Radius	Left	9	11	2	42	6	36
	Right	7	13	2	33	5	30
Ulna	Left	6	11	6	75	3	10
	Right	9	14	12	81	2	11
Humerus	Left	19	21	20	41	11	92
	Right	18	22	24	40	15	80
Tibia	Left	12	14	27	64	3	67
	Right	13	21	36	47	3	58
Femur	Left	16	32	51	114	25	78
	Right	12	43	50	74	22	76
Maximum Total of Individuals:		19	51	51	114	25	92

Minimum population required to provide the sample obtained:

Immature individuals: 51 fragmentary plus 16 entire femora = 67 immatures.

Adult individuals: 114 fragmentary plus 32 entire femora = 146 adults.

213 = Minimum Total Population Present in Sample.

the total of whole or fragmentary bones in 213, as stated previously, and is made up of 16 immature and 32 adult whole femora, and 51 immature and 114 adult fragmentary femora. In table 3 the ratio of males to females was 27 : 50 amongst all ages and based upon the recovered crania or cranial fragments. It can therefore be postulated that the same ratio probably held for the postcranial remains, and that they represent 75 males and 138 females respectively. If the allowance is

made for the absence of young adult males because of hunting accidents and tribal welfare, then a further number must be added to the male ratio. The addition of the 23 young adult males to the ratio in table 3 levels both the ratio for the 16-25 years group and the total ratio for all specimens. Therefore it may be suggested tentatively that a further 63 males are probably missing from the total recovered sample as represented by the postcranial material. The total deaths then would be 276 males and females for the period which the 213 recovered individuals represent, being composed of 138 males and 138 females approximately.

It is now possible to estimate the total number of deaths which occurred in the population during the estimated 10 to 15 year period which ended with the construction of the Tabor Hill ossuaries. For this calculation, the areas of the ossuaries are assumed to be proportional to the number of individuals buried within them. The areas for the large and small ossuaries were found to be 402.5 sq ft and 40.9 sq ft respectively, giving a total area of 443.4 sq ft. Volumes are not used, as the thickness of the stratum of bones in the two ossuaries is about equal. Since about half of the larger ossuary and four fifths of the smaller ossuary only were salvaged, the portions represent an estimated 276 deaths, giving the proportion of 233.97 sq ft for 276 deaths. When this is adjusted to 443.4 sq ft, the total number of deaths becomes 523.

Estimate of Regional Population.

In the previous section, it was reported that the remains of 213 individuals were actually recovered. A further 63 males are considered to be absent from the sample. When the male to female ratio is made equal by the addition of these 63 males, a total of 276 is obtained for the actual number of deaths represented by the 213 individuals recovered. Then by simple proportion, an estimate was made of the total number of deaths which the whole of both ossuaries would represent had they been undamaged and completely excavated, giving a final figure of 523. This figure of 523 would include all the burials within the ossuaries, whether recovered or not, as well as the estimated number of males between the ages of 16 and 25 years whose death can be attributed to hunting accidents and warfare, and whose bodies were never recovered.

An attempt to estimate the regional population can now be made. It is known that the Iroquois occupied their villages for periods of from 10 to 15 years. Therefore the average number of deaths per year can be estimated as lying between 34.8 and 52.3.

Hooton (1920) suggests an annual death rate of 2.3 per hundred

as a reasonable average rate for his Madisonville cemetery, and rounds this figure to 3.0 per hundred to account for infant burials which might have been overlooked or unrecorded. He bases these figures on a comparison of the death rates of various age groups in different European countries, and found that the death rate for Switzerland in 1873-77 agreed most closely with the recorded deathrate of the Madisonville cemetery (table 5). If similar figures are calculated for the Tabor Hill

TABLE 5

Comparison of Average Annual Deathrates Per Hundred for Two European Countries and Two American Indian Cemeteries. The Tabor Hill Figures are Based upon Postcranial Material (See Table 2), after Hooton (1937)

PLACE	YEARS COVERED	PERCENTAGE DEATHS AT AGES 0–10	10–20	20—	10—	MEAN ANNUAL DEATHRATE
France	1866–1877	32.28	4.25	63.47	67.62	2.46
Switzerland	1873–1877	36.94	3.72	59.33	63.05	2.38
Madisonville	100 year period	35.2	3.70	61.1	64.80	2.38 (3.00)
Tabor Hill	10–15 year period	31.5			68.5	2.46 (3.00)

TABLE 6

Estimations of Populations Living During the Period Represented by the Tabor Hill Ossuaries, Based on the Methods of Hooton (1920).

DEATHS PER YEAR	34.8	52.3
DEATH RATES PER HUNDRED	ESTIMATED POPULATIONS	
3.00	1,160	1,743
2.46	1,415	2,126

ossuaries, and a similar comparison is made with Hooton's table, then a death rate of 2.46 per hundred is suggested, similar to that for France in the period 1866-77. Accepting these figures of an annual death rate of 2.46 or 3.0 per hundred, and 34.8 to 53.4 deaths per year, then the group which used Tabor Hill as a burial ground must have numbered between 1,160 and 2,126 people (table 6).

The small number of casualties from warfare and hunting accidents is striking when the estimated loss of young males is compared with the population estimates. If we add the 23 missing males to the total known deaths in table 3, we find that 20.3 of every 100 deaths can be attributed

to either accident or warfare. Of the estimated 523 deaths over the 10 to 15 year period, 106.2 can be attributed to the same causes. The casualty rate per annum will therefore be between 7.1 and 10.6 on total deaths of between 34.8 and 52.3, or approximately 20.4% per annum. From these can be estimated the casualty rates as percentages of the estimated populations (table 7). These low casualty rates from warfare, etc., may indicate that the population was not involved in continuous fighting, but was mainly concerned with the struggle to produce food and to keep warm.

TABLE 7

Death Rates for the Tabor Hill Population Expressed as Percentages of the Estimated Populations.

TIME IN YEARS	ESTIMATED TOTAL DEATHS PER ANNUM	ESTIMATED TOTAL CASUALTIES PER ANNUM	CASUALTIES EXPRESSED AS: % TOTAL DEATHS	% OF ESTIMATED POPULATION POP. 1160	POP. 2126
10(min.)	52.3	10.6	20.4	0.92	0.45
15(max.)	34.8	7.1	20.4	0.61	0.33

Further methods of estimating the population are available. Kidder (1958) and Hooton (1930) working on the skeletal remains of the Pecos Pueblo population give figures of 38.92% and 39.8% respectively for deaths of individuals at less than 20 years of age. The reported deaths from Tabor Hill for this age component based on cranial remains are 38 out of 90, or 42.2%; with the addition of individuals not recovered because of hunting accidents or warfare, the figures become 52 out of 113, or 46.0%. If we equate the under-20 year component with the 'immature' group of individuals based on the post-cranial remains, then the figures are 67 out of 146, or 45.89%; and with the addition of the individuals absent because of hunting accidents or warfare, a further 63 individuals, the figures become 102 out of 174, or 58.56%. Howells (1959) suggests that a deathrate for the under-20 component should be close to 60% of the total deathrate. The last estimate for Tabor Hill is in close agreement with Howells' revised figure. Agreement, or at least support, is therefore available for our suggestions as to the number of individuals absent from 16-25 year groups from the Tabor Hill sample.

Goldstein (1953) working on Indian skeletal remains from "all major regions of the state" of Texas and representing "peoples living

between 800-1700 A. D." gives a figure of 27.6% for deaths of individuals at less than 20 years of age. This is lower than the figures ranging from 38.92% to 60% given above. Goldstein suggests that the greater likelihood of loss or disintegration of the fragile infant skeletons accounts for the low proportion of deaths in this age grouping. The presence in his sample of both isolated burials and burials comprising a population unit would tend to show a low proportion of child burials, as the isolated child burial would have small chance of preservation and fortuitous discovery. The inclusion of the isolated burials, the large area from which the sample is drawn, and the wide spread in time all militate against a just comparison of Goldstein's figures and those from Pecos Pueblo or Tabor Hill.

A comparison of the Tabor Hill deathrates was made with those from Indian Knoll, as reported by Snow (1948). Howells has computed an age distribution of the 1132 skeletons from Indian Knoll and has rounded this off to give a rough life table. In this age distribution, deaths under 21 years are 57%, which approximates very closely to the figure for Tabor Hill (58.56%) and to Pecos (60% assumed). However, the distributions of both samples do not follow the same age curve. Both curves are bimodal, but the maxima fall at 4 and 34 years for Indian Knoll and 20 and 45 years for Tabor Hill. The presence of a 10-15 year discrepancy could be attributed to differing occupations or diets, although these are probably not the only sources of difference. It might be concluded that the causes of death in the two populations were different, because the majority of deaths occurred at different times in the life-spans of the two populations. Such a difference could be an epidemic which might increase the deaths in a certain age group, or a series of mild winters and good years which might temporarily reduce the deathrates. However, it is obvious that both populations had a high mortality and a generally short life. The average age at death of those reaching the age of 20 was 30.4 years for Indian Knoll and 30.35 years for Tabor Hill. Thus, with the exception of the disagreement between the two life-tables, the Indian Knoll and Tabor Hill populations seem to have been very similar in their general history. Since Indian Knoll was a pre-agricultural group and Tabor Hill an incipient agricultural population, their methods of subsistence might easily have been very similar, the main source of food being meat obtained by hunting, some maize, beans, or squash obtained by primitive agriculture, and additional food obtained by gathering.

The use of a deathrate of 3% per annum is suggested by Howells

as being too low, and he favours one nearer 5% for the Indian Knoll population. We are fortunate at Tabor Hill in knowing that the Iroquois, at least in the later period, practiced a shifting economy, and that each site was probably occupied for only 10-15 years. This has already allowed us to estimate the maximum population at 2,126 and the minimum at 1,160 (table 6). We may finally summarise the population figures as being between 1,000 and 2,000 for Tabor Hill, and that the death rates were about 3% per annum, with about 57% of the deaths occurring under 20 years of age and an average life expectancy for those that passed 20 years of age of a further 10 years, the average adult death taking place at 30.35 years.

Sexual Variation in the Postcranial Skeletal Remains.

It is not proposed here to illustrate the distribution of all the osteometric data; but it is desirable to mention that in all cases such distribution showed marked bimodal curves. These curves almost entirely corresponded to the male and female specimens from the samples. As an example of these, the distribution of the diameters of the femur heads is given in figure 3.

From figure 3 it can be seen that the maxima of the bimodal curve showing the distribution of magnitudes of the femur head diameters for the 245 specimens correspond with the maxima for the distribution of the identified 28 male and 33 female femur heads. The 2 mm running average (dot-dashed line) smooths the distribution, emphasising its bimodal nature, and the maxima so produced coincide almost exactly with those for both male and female distributions. It is therefore reasonable to conclude that in all the bimodal curves showing the distribution for the various measurements, the two maxima reflect male and female variation in the absolute measurements of the skeleton. Table 8 gives the means and the numbers of various measurements of specimens of longbones by sex and side of the body.

STATURE OF THE TABOR HILL INDIANS.

Pearson and Bell's (1919) formulae for calculating the stature of individuals from the length of one or more longbones were applied to the various longbones available. The results of these calculations are given in table 9. The male statures range from 1657 to 1735 mm, and

TABLE 8

Means and Numbers of Measurements for Various Dimensions of the Long Bones from the Tabor Hill Ossuaries, Separated into Groups by Sex and Side of the Body. The statures Which Can be Calculated from These Means are given in the Right-Hand Column. (Standard Deviations and Standard Errors have not been given as in the Majority of Samples the number of Specimens Available Fall Below 15. This is True for all the Measurements Taken on all bones, and, consequently Means and Number of Speciments only are given).

MEAN MEASUREMENTS OF LONG-BONES.

BONE		BICONDYLAR LENGTH	MAXIMUM LENGTH	LENGTH WITH SPINE	MINOR DIAMETER	MAJOR DIAMETER	HEAD DIAMETER	PLATYKNEMIC INDEX	STATURE MM.
Males									
Radius—	Left		262.6(5)						1718
	Right		273.5(8)						1753
Humerus—	Left		327.0(8)		15.5(8)	22.5(8)			1652
	Right		332.1(12)		16.4(12)	22.8(12)			1667
Tibia—	Left	395.8(8)	383.5(5)	400.0(6)	21.8(6)	37.6(6)		57.9	1727
	Right	393.8(8)	400.1(8)	401.6(8)	22.0(8)	39.8(8)		55.2	1722
Femur—	Left	446.0(12)	450.8(12)		27.0(12)	29.1(12)	46.5(12)		1651
	Right	450.9(17)	451.4(15)		26.6(17)	29.0(17)	47.3(16)		1660
Females									
Radius—	Left		248.8(6)						1643
	Right		254.5(7)						1663
Humerus—	Left		303.2(10)		14.1(15)	20.1(15)			1549
	Right		311.0(10)		14.8(10)	20.9(10)			1571
Tibia—	Left	369.2(8)	371.3(8)	374.0(8)	20.2(8)	34.6(8)		58.6	1615
	Right	361.7(13)	368.2(13)	368.5(13)	20.0(13)	34.3(13)		58.4	1596
Femur—	Left	430.0(15)	433.6(12)		24.7(15)	26.7(15)	42.4(14)		1564
	Right	424.3(22)	429.0(22)		23.9(21)	25.4(21)	42.2(20)		1553

the female from 1559 to 1651 mm. It is interesting to note that the males exceed the females by approximately 80 mm for the tibiae and radii and 100 mm for the humeri and femora. Knowles remarks on the greater proportionate length of the tibia in the skeleton of Indians when compared with that of Europeans. The tibio-femoral index for the Roebuck Iroquois for example, is 85 for both males and females, in comparison with 82 in white males and slightly less for white females, (Hrdlička),

TABLE 9

Statures Calculated for Both Sexes from the Four Long Bones given, together with Numbers Available and Mean Lenth of the Bones. All Measurements in mm.

BONES	SEX	NUMBER	MEAN LENGTH MM.	STATURE MM.
Femora	Male	29	449	1657
	Female	37	427	1559
Humeri	Male	20	329	1658
	Female	25	307	1560
Tibiae	Male	14	394	1722
	Female	21	380	1641
Radii	Male	13	268	1735
	Female	13	251	1651

1952). This proportionately larger tibia is reflected in the larger stature calculated from the tibiae. Knowles also shows that the humero-femoral index of the Roebuck Iroquois is " very similar to the humero-femoral index in various white groups." However, stature may be slightly underestimated is derived from measurements on the femur and humerus. Therefore a more reliable figure may be obtained by comparing statures derived from the femora, humeri, radii and tibiae, with statures given for other Iroquois groups.

Table 10 gives statures in mm for two recent populations of Iroquois Indians, those from the Six Nations Reserve, Brant County, Ontario, and the Tonawanda Reserve, New York State (Knowles, 1911 and 1915, cited in Knowles, 1937), and two archaeological sites, the Roebuck Site, Grenville County, Ontario (Knowles, 1937) and the Tabor Hill Ossuary, York County, Ontario. The median and mean statures as derived from table 9 are included. Marked agreement between all the statures for the various male populations is shown, the stature for the Tabor Hill popu-

lation lying within the limits of the two recent populations and the Roebuck sample. The female statures for Tabor Hill are higher by some thirty or forty millemetres than the others, the only explanation that can be offered, except normal population variation, is that there is a bias towards small male size because the larger males are represented in the absent 16-25 year group.

The Tabor Hill population is therefore slightly greater in stature than any of the other three Indian populations, equalling in stature that of the Welsh (1695 mm), or French Canadian Soldiers (1703 mm), and being slightly smaller than the English (1712 mm) (Deniker, 1906).

TABLE 10

Statures Calculated from the Long Bones of the Various Iroquois Indian Populations given, Together with an Average Stature Derived from All Populations. All measurements in mm. (Data from Knowles, 1937).

SEX	ROEBUCK	SIX NATIONS	TONAWANDA	TABOR HILL MEDIAN	TABOR HILL MEAN
Male	1687	1705	1688	1698	1693
Female	1562	1571	1559	1605	1603

COMPARISON OF TABOR HILL SKELETAL MATERIAL WITH OTHER IROQUOIS SKELETONS

1. *Postcranial Material.*

Table 11 gives the measurements for the postcranial material from the Roebuck Site, Middleport Ossuary, Peterborough County Mounds (Otonabee Mound, Serpent Mound, and Upper Miller Mounds combined (Knowles, 1937)), Younge Site (Greenman, 1937), and the new Tabor Hill material.

A comparison of the measurements given in this table shows that the Tabor Hill material agrees fairly well with that from other sites in Ontario. In both males and females the measurements for the three limb elements usually fall within the range covered by the measurements from the other four sites, although the males tend to be shorter and the females longer. This agrees with the figures for the calculated statures (table 10) where the female Tabor Hill population exceeds that of the other populations, while the male Tabor Hill population was indistinguishable.

TABLE 11

Mean Measurements of Long Bones from Tabor Hill Ossuaries and Four Other Indian Burial Sites; the Figures in Brackets Indicate the Number of Specimens.

		HUMERI MAX. LENGTH	FEMORA MAX. LENGTH	FEMORA OBLIQUE	TIBIAE SPINE INC.	TIBIAE SPINE EXC.	RADII MAX. LENGTH
Mean Lengths of Male Long-bones.							
Tabor Hill	Left	327.0(8)	450.8(12)	446.0(12)	400.5(6)	393.4(5)	262.6(5)
	Right	332.1(12)	451.4(15)	451.0(17)	401.6(8)	400.1(8)	273.5(8)
Roebuck	Left	332.5(2)	462.0(3)	458.7(3)	402.0(2)	395.5(2)	
	Right	338.0(2)	464.7(3)	461.0(3)	397.3(3)	392.3(3)	
Middleport	Left	331.6(11)	465.5(8)	462.4(8)	391.8(11)	387.5(11)	
	Right	331.1(11)	463.3(7)	459.1(7)	394.0(6)	389.2(6)	
Peterborough County Mounds	Left		474.5(10)		400.6(7)		
	Right		471.9(11)		397.8(10)		
Younge	Left	331.3(3)	458.7(12)		388.1(8)		L & R
	Right	332.3(7)	451.5(12)		395.5(6)		261.7(7)
Mean Lengths of Female Long-bones.							
Tabor Hill	Left	303.2(15)	433.6(12)	430.0(15)	374.0(8)	371.4(8)	248.8(6)
	Right	311.0(10)	429.0(22)	424.0(22)	368.5(13)	368.2(13)	254.5(6)
Roebuck	Left	302.4(21)	431.2(25)	422.7(18)	362.8(17)	357.9(13)	
	Right	305.8(24)	428.8(26)	433.1(17)	360.9(17)	355.2(9)	
Middleport	Left	307.0(5)	449.8(5)	443.2(5)	366.5(4)	361.8(4)	
	Right	302.8(4)	426.6(5)	418.0(5)	367.0(8)	360.5(8)	
Peterborough County Mounds	Left		428.0(3)		358.8(4)		
	Right	319.0(3)					
Younge	Left	311.8(6)	420.8(11)		348.6(8)		L & R
	Right	308.6(7)	418.2(11)		345.1(9)		236.3(11)

2. *Measurements of Crania.*

Table 12 gives comparative cranial measurements and indices for the Tabor Hill material, Roebuck Ossuary, Middleport Ossuary, Peterborough County Mounds, Huron Ossuaries, and the Younge Site. It can be seen from these sets of figures that the Tabor Hill populations had more brachycephalic crania than did the other populations. The male Tabor Hill population does not resemble the population of any other site, the similarities in measurements and indices being approximately evenly distributed throughout the other populations. The female Tabor Hill population, on the other hand, resembles most closely the Roebuck population in the shape of the cranium, and the Peterborough County Mounds populations in the nasal proportions. It is probable, therefore, that the population from Tabor Hill is merely a segment of the overall Iroquois population in Ontario. Such differences as exist from the other sample populations simply represent normal population variation because of the partial reproductive isolation that occurs in scattered populations.

Knowles (1937) states that the "prehistoric Iroquois of Ontario seem to have been composed of a dolichocephalic people mixed with a certain proportion of brachycephalic types; the brachycephalic element varied in amount among the different tribes and probably varied also at different periods of time."

In table 13 the distribution, numbers and means of the cephalic indices for the Tabor Hill, Nottawasaga, Roebuck, two Huron Ossuaries and the Younge Site are given. It is evident that the Tabor Hill population was definitely mesocephalic, with only one dolichocephalic individual and nine brachycephalic individuals. The Tabor Hill population therefore does not entirely fit into the general concept of Knowles' statement, since within his concept it must be considered to be one of the brachycephalic groups in which little or no dolichocephalic admixture had taken place.

If the mean cranial indices given by Knowles for the males and females for his sites are compared with the indices for the Tabor Hill population (table 14) then it can be seen that the conclusion that the Tabor Hill population is brachycephalic in comparison with the remainder of the Iroquois cephalic indices is supported, as only five of the 21 sample groups are equally or more brachycephalic than the Tabor Hill population.

It is reasonable to conclude that while the Tabor Hill sample is somewhat more brachycephalic than might be expected, it fits none-the-less well within the overall distribution of the range of the cephalic indices of the Iroquois tribes.

TABLE 12

Mean Measurements of Crania from Tabor Hill Ossuaries and Six Other Indian Burial Sites; the Figures in Brackets Indicate the Number of Specimens

SITE	TABOR HILL	ROEBUCK	MIDDLEPORT	PETERBORO' MOUNDS	5 HURON OSSUARIES	YOUNGE	KRIEGER
			Mean Cranial Measurements—Male				
Length	184.5(11)	188.7(3)	186.7(9)	188.1(15)	188.2(63)	183.3(21)	180.0(2)
Breadth	144.3(11)	147.0(3)	143.4(8)	142.8(16)	140.5(62)	138.0(19)	124.5(2)
Cranial Index	78.2(11)	78.0(3)	77.0(8)	76.1(15)	74.7(62)	75.2(19)	69.0(2)
Basibregmatic Height	136.2(7)	142.0(2)	137.6(7)	136.1(13)	139.7(46)		123.6(3)
Bizygomatic Breadth	140.6(6)	144.0(3)	145.7(6)	142.8(10)	141.4(39)	138.7(13)	119.3(3)
Upper Facial Height	74.1(7)	75.5(2)	75.8(6)	70.3(11)	73.8(28)	68.7(14)	67.0(3)
Nasal Height	53.5(6)	56.5(2)	56.0(7)	54.3(12)	55.5(32)	49.6(14)	50.0(3)
Nasal Width	29.1(7)	28.0(3)	27.0(7)	25.9(12)	27.3(32)	28.2(14)	23.3(3)
Nasal Index	50.2(6)	48.0(2)	49.5(7)	47.9(12)	48.5(32)	57.0(13)	47.0(3)
			Mean Cranial Measurements—Female				
Length	171.9(15)	178.6(25)	177.5(16)	183.8(5)	179.2(32)	177.2(13)	179.6(3)
Breadth	140.4(15)	138.8(25)	137.6(14)	136.4(5)	137.2(32)	134.5(13)	121.0(2)
Cranial Index	79.0(15)	77.9(24)	77.4(14)	75.6(5)	76.6(32)	75.9(13)	68.5(2)
Basibregmatic Height	131.2(14)	131.4(21)	132.9(14)	134.4(5)	133.3(26)		139.3(3)
Bizygomatic Breadth	133.5(4)	132.3(23)	130.5(11)	131.0(3)	129.8(17)	130.4(5)	104.0(1)
Upper Facial Height	69.6(3)	69.5(20)	67.2(9)	66.0(4)	67.6(12)	65.8(6)	60.0(1)
Nasal Height	51.3(3)	51.4(26)	50.2(10)	51.3(4)	50.7(14)	49.6(14)	43.0(1)
Nasal Width	24.7(3)	36.9(24)	26.1(10)	25.0(4)	26.6(15)	28.2(14)	21.0(1)
Nasal Index	48.1(3)	52.3(24)	52.2(10)	48.8(4)	52.4(14)	27.0(13)	51.0(1)

CONCLUSIONS

The population represented by the skeletal remains recovered from the Tabor Hill Ossuaries can be referred to the Iroquois on the physical

TABLE 13

Distribution and Means of the Cephalic Indices for the Tabor Hill Ossuaries' Crania Compared with Other Sites

(*Mainly from Knowles, 1937, and Greenman, 1937*).

		MALE						FEMALE					
	CEPHALIC INDEX	Tabor Hill	Nottawasaga	Innisfil Twp.	Flos Twp.	Roebuck	Younge Site	Tabor Hill	Nottawasaga	Innisfil Twp.	Flos Twp.	Roebuck	Younge Site
Dolichocephalic	68						1						
	69						1						
	70		1	1			1		1				1
	71			1	1		1						
	72	1					1						2
	73						3		1			1	1
	74			1		1	1			1		2	1
Mesocephalic	75		1	3			2	3				2	3
	76	1		2			2	2				2	
	77	4		1			1	2	1	1		4	1
	78	1		1			1					4	1
	79	1			1		3	3		1		2	1
	80					2						1	1
Brachycephalic	81							2				4	1
	82	1					1	1				2	
	83	2								1			
	84			1				1					
	85												
	86												
	87												
	88							2					
Total		11	2	11	2	3	19	16	3	4	0	24	13
Mean Index		78.2	72.5	75.6	74.5	78.0	75.2	79.0	73.3	78.0	—	77.9	75.9

evidence available, their stature being approximately 1695 mm for males and 1605 mm for females. The population was composed of 213 actual

remains, and when allowances are made for the incomplete excavation of the ossuaries and the possible loss of young adult males from warfare

TABLE 14

List of Mean Cranial Indices and Number of Specimens from some Indian Sites in Northeastern North America, Arranged According to Descending Magnitude. (1) Crania in Royal Ontario Museum, (2) Crania in Care of Buffalo Society of Natural Sciences. (Mainly from Knowles, 1937, also Hooton, 1920, and Greenman, 1937.)

	MALES		FEMALES	
SITE OR POPULATION	NO.	MEAN CRANIAL INDEX	NO.	MEAN CRANIAL INDEX
Clinton Twp., Lincoln Co.	1	80.0	—	—
Roebuck Site, Augusta Twp., Grenville Co.	3	80.0	24	79.9
Madisonville Site, Ohio	14	79.6	5	79.8
Tonawanda Reserve, New York State	39	79.6	43	79.4
Six Nations Reserve, Brant Co.	244	79.1	163	80.0
Tabor Hill Ossuaries, York Co.	*11*	*78.3*	*15*	*79.0*
Humberstone Twp., Welland Co. (2)	4	77.8	2	75.5
Huron	13	77.4	4	80.0
Beverly Twp., Wentworth Co.	5	77.2	3	71.7
Middleport, Onondaga Twp., Brant Co.	8	77.0	14	77.4
Huron and ? Huron	66	76.7	33	78.6
Markham Twp., York Co.	4	76.3	4	74.8
Manvers Twp., Durham Co.	19	76.0	10	76.6
Humberstone Twp., Welland Co. (1)	4	76.0	2	79.0
Inisfiel Twp., Simcoe Co.	11	75.6	4	78.0
Younge Site, Goodland Twp., Lapeer Co., Mich.	19	75.3	13	75.9
Sealey Farm, Brant Co.	1	75.0	—	—
Walker Farm, Brant Co.	1	75.0	—	—
Flos Twp., Simcoe Co.	2	74.5	—	—
Sherks, Humberstone Twp., Welland Co.	4	74.0	2	77.5
Port Colborne, Welland Co.	3	73.7	2	76.0
New York State	19	73.5	15	74.8
Nr. Clearville, Kent Co.	4	73.5	4	78.3
Vaughan Twp., York Co.	26	73.3	14	76.8
Grand Island, New York State	4	73.3	2	70.0
Orangeport, New York State	6	72.7	5	75.6
Nottawasaga Ossuary, Simcoe Co.	2	72.5	3	73.3
Jerseyville, Wentworth Co.	1	71.0	—	—
Krieger Woodland Site, Kent Co.	2	69.0	2	68.5

and hunting accidents an upper estimate of 523 individuals can be suggested as inclusive of all those members of the population that died

during the period represented by the ossuaries. When this figure is applied to death-rates, a regional population of some 1,160 to 2,126 individuals can be suggested. The population was mesocephalic to brachycephalic in cranial proportions, in which it differed slightly from the norm of the other Iroquois skeletal remains with which it was compared.

SUMMARY

1. Two ossuaries are reported from Tabor Hill, Scarborough Township, York County, Ontario. Both are Iroquois in origin, and approximately 700 years old (and lie within a sandy-loam soil typical of Southern Ontario).

2. A comparison of the skeletal elements shows that some 213 individuals can be counted with certainty, but since partial destruction of both ossuaries had taken place, an estimated number including the destroyed portions and absent individuals would be about 523 individuals.

3. The sex distribution of the crania was 27 males to 50 females, or 75 males to 138 females of the recovered sample. It is suggested that this differential in the sex ratio was caused by the failure of hunting or war parties to retrieve all of the bodies of those who met their deaths some distance from home.

4. Measurements of the post-cranial skeleton show a bimodal distribution. This can be correlated with the two sexes, reflecting the variation in size between the sexes in similar dimensions.

5. Stature is calculated for the population by sex, being 1689 mm for males and 1605 mm for females. These are greater than the statures given for the Roebuck Ossuary and many living Iroquois groups.

6. Comparison of post-cranial material shows the population to fall well within the normal distribution of Iroquois measurements.

7. Comparison of cranial measurements and Cranial Indices shows similar agreement, falling within the upper limits of variation for the cranial index of Iroquois Indians, although the population shows a more brachycephalic mean cranial index than the other ancient material with which it is compared.

8. The conclusion is drawn that these ossuaries show no marked divergence from the Iroquois pattern as regards either the skeletal elements or the method of burial.

LITERATURE CITED

DENIKER, J. 1906 Races of Man. Second edition. Appendix 1. Walter Scott. London.

GOLDSTEIN, M. S. 1953 Some vital statistics based on skeletal material. Human Biol., *25*: 3-10.

GREENMAN, E. F. 1937 The Younge Site. Occas. Contrib. Mus. Anthrop. Univ. Michigan, No. 6.

HOFFMANN, D. W. AND N. R. RICHARDS 1955 Soil Survey of York County. Rept. No. 19, Ontario Soil Surv., Expt. Farms Serv., Canada Dept. Agric. & Ontario Agric. Coll., Guelph.

HOOTON, E. A. 1920 Indian Village Site and Cemetery near Madisonville, Ohio. Pap. Peabody Mus, Amer. Archaeol. and Ethnol., Harvard Univ., volume 8.

—— 1930 The Indians of Pecos Pueblo. A study of their skeletal remains. Phillips Acad., Andover, Mass. Robert S. Peabody Foundation for Archaeology, Papers of the Southwestern Expedition, No. 4. New Haven, Connecticut.

HOWELLS, W. W. 1959 Estimating Population Numbers through Archaeological and Skeletal Remains. Viking Fund Publications in Anthropology (*in prep.*).

HRDLIČKA, A. 1916 Physical Anthropology of the Lenapes or Delawares, etc. Smithsonian Inst., Bur. Amer. Ethnol., Bull. 62.

—— 1952 Practical Anthropometry, Fourth edition. (Edited by T. D. Stewart), Wistar Inst. Anat. Biol., Philadelphia.

KIDD, K. E. 1953 The excavation and historical identification of a Huron ossuary. Amer. Antiq., *18*: 359-379.

—— 1956 A brief study of the human remains from the Krieger Woodland Site in Southwestern Ontario. Pennsylvania Archaeol., *26*: 15-26.

KIDDER, A. V. 1958 Pecos, New Mexico: archaeological notes. Phillips Acad., Andover, Mass. Papers of Robert S. Peabody Foundation for Archaeology, volume 5.

KNOWLES, SIR FRANCIS H. S. 1937 Physical Anthropology of the Roebuck Iroquois. Canada Dept. Mines and Resources, Nat. Mus. Canada, Bull. 87, Anthrop. Ser. No. 22.

KROGMAN, W. M. 1946 The skeleton in forensic medicine. Proc. Int. Med. Chicago, *16*: 154-167.

SNOW, C. E. 1948 Indian Knoll Skeletons of Site Ch. 2. Univ. Kentucky Repts. in Anthrop., *4*: 371-545.

PEARSON, K. AND J. BELL 1919 A study of the Longbones of the English Skeleton. Draper's Company Res. Mem., Biometric Ser. 10, Cambridge Univ. Press.

WINTEMBERG, W. J. 1936 Roebuck Prehistoric Village Site, Grenville Co., Ont. Canada Dept. Mines, Bull. 83, Anthrop. Ser. No. 19.

FACTORS ASSOCIATED WITH VARIATIONS IN MATERNAL DEATH RATES*

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ALTHOUGH the most important factors tending to increase the risk of death from deliveries and complications of pregnancy, childbirth, and the puerperium, and the means by which it is possible to decrease this risk, are becoming increasingly well understood, no serious attempt has been made to measure the impact of these influences in any precise way. As a result of a relatively cursory study arising from an attempt to measure the overall impact of hospital licensing by the states, the present writer has become convinced that the measurement of the most important influences on maternal death rates is entirely possible even with already existing data, and that the results could be of substantial value.

The focus of attention in the study reported here was to find variables having some direct connection with public policy, variations which might be associated with variations in maternal death rates, and where such covariation could reasonably be interpreted as causative. In order properly and convincingly to isolate the causative influence of such variables, it would be necessary to take into account the simultaneous influences of other important variables beyond policy control, or to eliminate such other influences. One way of doing this could be to undertake a controlled, laboratory-type experiment, in which all other variables than the ones directly studied were held constant. However, such a controlled experiment, while in principle possible, was quite out of the question in this case. Instead, the only feasible approach was to use multiple correlation techniques, where the disturbing influences of the more important non-policy variables could be "controlled" by their explicit simultaneous inclusion in the correlation-regression analysis.

The most important deficiency of this latter approach is that it can

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do nothing with variables which in a controlled experiment could be held constant but which cannot be (or have not been) measured. A special case of this difficulty would be that in which the actual observed variations in the "policy" variables, along with the variations in maternal deaths, were partly the result of a common outside (unobserved or unmeasured) cause. In this case the observed covariation would be a mixture of the true causative relation, if any, and of the covariation imposed by the disturbing outside influence. On the other hand, multiple correlation techniques have the advantage of measuring simultaneously the separate influences of all the included variables, including the "control" variables. This technique, taking due account of the covariation among the various influences studied, provides a measure of the influence of variations in each one with the others remaining constant.[1] In controlled experimentation this tends to be practicable only to a much more limited extent.

In the last resort the results of multiple correlation analysis, particularly when used in as subtle a problem as the analysis of factors affecting maternal death rates, can be fully trusted only if supplemented and reinforced by true controlled experimentation or other independent evidence. The risk that its results will be seriously misleading may be materially reduced, however, if various available devices are used to eliminate or take account of extraneous influences. For example, while a particular extraneous influence may not be directly measurable, it may be possible to find some measurable variable that is closely correlated with it, and which therefore may serve as a proxy variable which will largely eliminate (account for) the extraneous influence in question. Also, the study of movements over time, and of simultaneous cross-sectional data, may provide independent estimates of the effects of the same influences.

The data employed in this study consist primarily of state-by-state mortality and related statistics supplied in the *Vital Statistics of the United States.* Various income and population statistics were obtained from official publications of the U. S. Department of Commerce; and some data relating to hospitals were obtained from the American Hospital Association. The variables employed, and the sources from which they were obtained, are listed in detail in table 1.

In the 1955 cross sectional analysis the dependent variable used was

[1] Procedures and examples of this method are are given in Hoel (1947), *Introduction to Mathematical Statistics.* Wiley, London, chapters V and VII. See also Ezebial, M. (1941), *Methods of Correlation Analysis.* Wiley, New York.

TABLE 1

Variables Employed
(by States and the District of Columbia)

X_0: Maternal deaths per 10,000 live births, 1954-55. Source: *Vital Statistics of the United States*, 1955.

X_1: % of births attended by physician in hospital, 1955. Source: same as X_0.

X_2: % of population non-white, 1950. Source: *Statistical Abstract of the United States.*

X_3: Obstetrical beds per 1000 population, 1955. Source: Numbers of obstetrical beds from *Hospitals Guide*, 1955 (American Hospital Association).

X_4: Intensity of hospital licensing activity, 1955. Budget expenditures on all aspects of hospital licensing by states, divided by index of workload. Index of workload obtained as weighted sum of number of hospitals and number of active Hill-Burton projects, the latter receiving a weight of approximately 21 hospitals. Sources: Data on budgets, hospitals and weights obtained by questionaire survey of hospital licensing agencies in the states. Mimeographed data on numbers of Hill-Burton projects, and supplementary data on hospitals supplied by U. S. Public Health Service.

X_5: Income per capita, 1955. Source: *Personal Income by States since 1929*, supplement to the *Survey of Current Business*, U. S. Department of Commerce (Washington, 1956).

X_6: % of population in urban places, 1950. Source: same as X_2.

X_7: Index of stringency of state regulations for maternity wards, 1955. Source: Regulations supplied by the licensing agencies. Index formed by a scoring system for inclusion, partial inclusion, or omission of various typical regulations, weighed roughly according to expert opinion on their relative importance.

X_8: X_4 raised to the power X_7. To allow for possible interaction between X_4 and X_7.

X_9: Per capita federal grants-in-aid for maternal and child health programs. Source: Grants-in-aid data from *Federal Grants-in-Aid to States and Payments to Individuals.* Report of the Joint Committee on Reduction of Nonessential Federal Expenditures, Congress of the U. S. (Washington, 1957).

X_{10}: Per cent of eligible hospitals accredited by the Joint Commission for Accreditation of Hospitals, 1955. Temporary accreditations given a value of 1/2. Eligible hospitals counted as all hospitals over 25 beds listed by the American Hospital Association. Source: data supplied by American Hospital Association.

ΔX_0: Difference between X_0 and the corresponding figure for 1951-52.

ΔX_1: Difference between X_1 and the corresponding figure for 1952.

maternal deaths per 10,000 live births for the two years 1954-55. This was suggested to me by the Public Health Service, who have gone entirely to two-year average reporting on this statistic in order to reduce the influence of year to year random variation, particularly in the smaller states. The independent "causal" variables employed generally refer to 1955, mainly for the sake of simplicity, although it would have been more appropriate to use a 1954-55 average for these also; this makes very little difference because the year-to-year changes in these statistics are small relative to state-to-state variation. In the analysis of changes in maternal death rates from 1951-52 to 1954-55 the only independent variable that could be used was per cent of births in hospitals, because other relevant variables are not available on a year-to-year basis (or could not be obtained with the time and resources available).

The most pertinent results are presented in table 2. The first set of results represent perhaps the more reliable estimates of the various influences. For each of the regressions numbered 1, 2, 3 the first number appearing in the column corresponding to each variable is the estimated effect (coefficient) of that variable; the second number, appearing in parentheses, is the standard error of the coefficient. The last column in table 2 goves the coefficients of determination of the regressions. Even the lowest of them in this Table is statistically significant.

Regressions 1 and 2 differ by the omission from the first of the variable X_3, obstetrical beds per 1000 population; both omit the variable X_4. The variable X_3 was included in the hope that it would measure the extent of the practice of segregating obstetrical patients from other patients in hospitals, since "obstetrical beds" reported to the A. H. A. are presumably beds specifically allocated to obstetrical patients and therefore in most cases segregated from other patients. Insofar as this practice reduces the danger of infection after childbirth, it should be reflected in lower death rates. X_3 falls short of conventional levels of statistical significance, since a result this strong could occur by chance more han 10% of the time if no relation were in fact present. It is strong enough to be suggestive, however, and does give hope that further investigation with more and better data might very likely yield conclusive results.

A result not shown in the table is that the average expected maternal death rate in an all-white state if no births occurred in hospitals would be around 24 per 100,000 live births. The coefficient for births in hospital obtained from the second regression implies that if all births were in hospital this rate would be reduced by 19.5 to around 4.5 per 10,000 live births, *i. e.*, that it is less than one-fifth as risky to have a birth in hospital as to have it at home or elsewhere. In the light of the qualifying

TABLE 2

	X_1: % OF BIRTHS IN HOSPITALS	X_2: % OF POPULATION NON-WHITE	X_3: OBSTETRICAL BEDS PER 1000 POPULATION	X_4: RELATIVE INTENSITY OF HOSPITAL LICENSING ACTIVITY	R^2
A. Basic Regressions: Maternal Death Rates 1954-55					
1) Coefficient	—.213	.062	—	—	.872
(Standard Error of Coefficient)	(.028)	(.019)	—	—	
2)	—.195	.058	—2.089	—	.879
	(.029)	(.019)	(1.354)	—	
3)	—.219	.060	—	.0021	.876
	(.029)	(.020)	—	.0025	
4)	—.284	—	—	—	.844
	(.018)				
B. *Check Regressions: Maternal Death Rates 1954-55*					
4a) Low-Mortality States	—.013	.076	—1.331		.158
	(.189)	(.071)	(1.560)		
4b) High Mortality States	—.207	.039	—.327		.848
	(.042)	(.025)	(2.949)		

	INDEPENDENT CHANGE	ΔX_1: CHANGE IN % OF BIRTHS IN HOSPITAL	R^2
C. *Check Regressions: Changes in Maternal Death Rates 1951-52 to 1954-55*			
5a) All States	—1.226	—.413	.445
		(.067)	
5b) Low-Mortality States	—.68	—1.162	.223
		(.452)	
5c) High-Mortality States	—1.358	—.382	.423
		(.095)	

remarks that have been made above, however, this result must be interpreted with caution. It is entirely possible and indeed plausible, for instance, that the general level of obstetrical technology and safety is positively associated with the proportion of births occurring in hospitals, *i. e.*, that those states with the highest proportion of births in hospitals happen also to be those states in which new methods and standards have most rapidly and completely been adopted. In that case the coefficient of X_1 would show the influence of both these factors, and hence would exaggerate the influence of the choice of whether or not to have a birth in hospital. Similarly, the quality of diet, personal hygiene, and adequacy of pre-natal care of prospective mothers might happen to be positively associated with the per cent of births in hospitals. In this connection, it is noteworthy that the inclusion of X_3 in regression 2 had the effect of reducing (though not significantly) the estimated effect of X_1.

On the other hand, a considerable effort has been made to isolate the pure effect of birth in hospital. The race factor X_2, which is statistically significant, can most plausibly be interpreted as showing the effect of the poorer diet, hygiene, and pre-natal care which non-whites generally have. If X_3, obstetrical beds, correctly reflects segregation of obstetrical cases it should be highly correlated with the general state of medical technology, and possibly also with the size of the hospital. Income per capita, which ought to be highly correlated with these "extraneous" influences, was tested and found to be quite insignificant and to have no appreciable effect on the estimated coefficient for births in hospitals. Similarly the size factor, as measured by average assets per hospital, was found to be insignificant and to have no appreciable effect on the coefficients of X_1 and X_3. I am inclined to conjecture, therefore, that when further and more detailed research is done on this question, the independent effect of birth in hospital on the risk of death in childbirth will be found to be only a little smaller, if at all, than the estimate obtained here.

Regression 3 represents an attempt to find the effect of state regulation of hospitals. As maternity wards are a small part of the state agencies' responsibilities, however, and as X_4 is a crude measure of their activities, it is not surprising that no significant effect was found. Other attempts to measure the governmental impact on maternal death rates were equally unsuccessful, as indicated in table 3 (Appendix). Regression 4, which omits all variables except X_1, shows the importance omitted factors can have. This regression's coefficient for X_1 is significantly higher than the others, erroneously reflecting in part the influences of X_2 and X_3.

Besides the use of other variables, as indicated in the above discussion and in table 1, certain other devices are available to check on the validity of the basic regressions. One is to split the sample more or less logically into halves and to see if the results within the two subsamples thus obtained are broadly consistent with the results for the whole sample. The means of division that was employed in this case was to take the 25 states with the lowest maternal death rates as one subsample and the remaining 23 states and D. C. as the other subsample.

At first glance, this has quite striking effects on the coefficients, particularly on those for X_1 and X_3, as seen in regressions 4a and 4b of table 2. However, the observed variations between the two samples of all the coefficients, relative to their standard errors, are well within the limits of what may be expected from pure chance variation. This is especially evident in the cases of X_2 and X_3.

Some special interest, however, attaches to the effect of subdivision of the sample on the coefficient of X_1, per cent of births in hospitals. In the low-mortality states this coefficient nearly vanishes, and its standard error becomes very large. The explanation for this is that in 1955 the per cent of births in hospitals was virtually identical in the low-mortality states at around 99.5% and over, *i. e.*, nearly 100%. A factor which does not vary cannot of course explain any of the variation in anything else. Owing to this the result of regression 4a is entirely within the bounds of variation reasonably attributable to chance. (This also explains the low R^2 for this regression.) Regression 4b, for the high-mortality states, is in general in closer conformity with regression 2, as within this subsample the variation in births in hospitals was about the same as that in the whole sample.

The statistical accident in the low mortality group was in fact advantageous for this study, as the lack of variation in the main explanatory variable provided an opportunity to look for the effects of other variables which otherwise might be masked by and confounded with this variable. The most promising hypothesis that seemed worth testing in this connection was that diet, hygiene, and so on would be a function of per capita income, and that a statistically significant coefficient for per capita income might therefore be found. As is indicated in the appendix, this was found not to be the case, either for all states or for either subgroup. Since the birth in hospital variable was virtually constant in the low mortality group of states, while per capita incomes vary considerably, the situation resembled one of experimental control, and income should show its full independent effect. The failure of per capita

incomes in this group is fairly conclusive evidence that variations in nutrition and hygiene, if important influences on maternal deaths, are uncorrelated with income; and this makes it less plausible to suppose that they are correlated with the per cent of births in hospitals. It therefore lends additional support to the belief that the bias in the estimate of the effect of birth in hospital is not likely to be serious.

A second, almost independent check on the basic results is provided by the third set of regressions in table 2. These are simple regressions (one explanatory variable) of the change from 1951-52 to 1954-55 in maternal death rates on the change from 1952 to 1955 in the per cent of births in hospitals. The "variable" labeled "Independent Change," that is the constant term, is an estimate of the amount by which maternal mortality rates would on the average have changed if the per cent of births in hospitals had not changed at all. That is, it is a measure of the degree of medical advance, as reflected in maternal death rates for given proportions of births in hospitals.

Regression 5a is for all states and D. C. together. If its results were to support the basic regression results, its coefficient for changes in X_1 would have to be substantially the same as the coefficient of X_1 in a comparable basic regression. As Regression 5a could only be carried out using changes in X_1, no data being available on changes in other factors, the most comparable basic regression is Regression 4. It can be seen that the coefficients do differ substantially, although the difference is just short of statistical significance.

Although various interpretations of this result are possible, the most plausible is that Regression 5a overstates the effect of birth in hospital even more than Regression 4, because of positive association between changes in per cent of births in hospitals and the rate of medical advance. That is, it seems likely that the states in which the per cent of births in hospitals went up the most between 1952 and 1955 were also those in which the rate of improvement in medical techniques and so on was the highest. In this case the regressions of changes in mortality rates on changes in per cent of births in hospitals would tend to exaggerate the influence of birth in hospital as such. Since the states with high mortality rates were those which in general had the greatest reductions in these rates and the greatest increases in per cent of births in hospitals, this would among other things imply that the high mortality states were tending to advance more rapidly in technology (for example because they had more ground to make up).

Regressions 5b and 5c add little that is useful in this context, although they cast some doubt on the uniformity of the relationship between the

subsamples. As in the case of regression 4a, the standard error of the coefficient of changes in per cent births in hospitals became very large in regression 5b because of relatively small variation of this factor in this subsample. Even with this large standard error, however, the coefficient is sufficiently out of line with the others to cast doubt on whether it differed purely by chance. (The difference is not statistically significant, but comes near to being so.) If it was not by chance, this would imply that the correlation between changes in births in hospitals and changes in omitted factors was higher in the advanced, low mortality states, than in the others.

Assuming that the interpretation offered here for the divergence between the results of regression 5a and the basic regressions is valid, the estimate of the coefficient of births in hospitals from, say, regression 2 may be used in conjunction with the data on changes in mortality rates and in births in hospitals to obtain better estimates of the independent changes in mortality rates. For what they are worth, these revised estimates of the average reduction in maternal death rates that would have occurred had there been no change in the per cent of births in hospitals are as follows:

All states	—1.793	per 10,000 live births
Low mortality states	—1.356	"
High mortality states	—2.207	"

Certain next steps follow more or less directly from what has been done here. The most obvious one is to make more extensive use of the year by year data in *Vital Statistics*, going back as far as it goes and repeating the regressions used here. However, the opportunities even with existing data are much greater than this. Mortality and birth in hospital data are reported annually by counties cross-classified by race. With these data something approaching a true controlled experiment would be possible, by taking matching pairs of counties identical in every possible respect (such as race composition, income levels, proportion of population rural, and so on) except for the measurable factors selected for special attention. The extent to which obstetrical patients are segregated from other patients is one especially interesting such factor, particularly from the standpoint of policy; but the influence of birth in hospital also merits further study.

APPENDIX

One of the problems in multiple correlation analysis concerns the precise form of the relationship between the variables. Some arbitrary

choice must be made on this matter, although experimentation with drastically different forms will usually suggest which of the forms tried fits the data (approximately) the best. When the fit is very poor, the choice of form is not likely to make much difference; but if the investigator has good reason to suppose one form is superior to another on analytical grounds, then he may expect that it will give the better fit.

The question of the form of the relationship is especially troublesome in the case of maternal death rates because it is quite likely that the form differs for different explanatory variables. The effect of birth in hospital may quite plausibly be supposed to be constant for different values of the variable; *i. e.*, it is reasonable to suppose that having a birth in hospital reduces the risk of death by the same absolute amount regardless of whether the going percentage of births in hospitals is 50% or 99%, if other things are equal. The fact that other factors are not generally equal may affect this, and may involve interaction effects; it is also conceivable that a high percentage of births in hospitals would involve crowding of facilities and therefore a reduction in the benefit of hospitalization.

Interaction effects are unlikely to be significant unless more than one variable can be identified as having a substantial explanatory value. Since one variable in this study did nearly all of the explaining, the others having only marginal effects if any, interaction may safely be ignored. This is fortunate, because the procedures for checking for possible interaction are exceedingly clumsy in multiple correlation analysis; an example of one possible such procedure is the use of the variable X_8 (see table 1).

Regardless of the form of the relationship between maternal death rates and per cent of births in hospitals, the constant proportional or logarithmic relationship is more plausible than a constant arithmetic (linear) relationship in the case of most other variables. For example, if it were true that per capita income changes were associated with changes in maternal death rates because of the former's association with such factors as diet, hygiene, and so on, it would be difficult to believe that a given change in income would produce even approximately the same numerical change in maternal death rates whatever their level. One would expect the effect to be much larger where the rates are high than where they are already low. In this case the logarithmic relationship, though not necessarily the correct one, is likely to be the better approximation.

Therefore the income variable and other such variables can be tested

for significance in the same regressions in which the goodness of fit of the logarithmic form is tested for per cent of births in hospitals. A few of the most interesting of these regressions, among the many tried, are shown in table 3 and table 4.

TABLE 3

Explanatory Variables *

Regression No.	$\log X_1$	$\log X_2$	$\log X_3$	$\log X_4$	$\log X_5$	$\log X_6$	$\log X_7$	$\log X_8$	$\log X_9$	$\log X_{10}$	R^2
6	—2.481		—.557	—.015	—.232	.202					.717
	(.767)		(.212)	(.075)	(.346)	(.214)					
7a	—.734		—.341	.170	—.065						.162
	(7.614)		(.345)	(.178)	(.415)						
7b	—2.322		—.186	.036	.024						.810
	(.560)		(.215)	(.058)	(.218)						
8	—2.247	.114	—.247	—.005							.765
	(.515)	(.037)	(.214)	.064							
9	—2.468	.140					—.069				.763
	(.556)	(.038)					(.114)				
10	—2.340	.147						—.011	.068		.767
	(.600)	(.040)						(.053)	(.087)		
11	—2.576		—.606							.041	.711
	(.598)		(.198)							(.124)	
12	—2.251	.114	—.247								.765
	(.508)	(.037)	(.211)								

* For descriptions of the variables, see table 1.

TABLE 4

	$\Delta \log X_1$	R^2
13a	—1.181	.029
13b	—21.639	.178
13c	—1.461	.101

Regression 6 represents an attempt to test income per capita and the urban-rural factor. Neither was significant, or even promising. Since this result was very unexpected in the case of per capita income, and it was thought that it might be due to an imperfect separation of this factor's influence from the influence of the factor per cent of births in hospitals, the device was employed of breaking the observations in two subsamples. As mentioned in the text, the very small variation of per cent of births in hospitals in the low-mortality states causes this variable to lose significance and gives per capita income a chance to show its independent effect. As can be seen in regression 7a, this failed to produce a significant coefficient for per capita income.

Regression 8 introduces the variable per cent of population non-white, which causes X_3, obstetrical beds, to lose significance. (Compare with regression 6.) This is due to an association between these two variables, the states with low per cents non-white tending to be those with relatively high availabilities of obstetrical beds; hence with per cent non-white omitted the other variable tended misleadingly to show both influences together in its coefficient. This gives a striking illustration of this difficulty, discussed in the main part of this article, which can arise in correlation analysis (or indeed in an imperfectly controlled experiment.)

Regressions 9 and 10 test additional policy variables, none of which proves significant. Regression 11 was undertaken partly because hospital accreditations, as a rough measure of the standards of medical care, might be viewed as a potentially effective policy variable analogous to the intensity of the licensing activity. It was also thought that it might uncover a possible spurious or misleading element in the relationship between births in hospitals and mortality rates. It is conceivable that a low mortality rate and a high per cent of births in hospitals might in part be the common result of a high level of health consciousness, where this health consciousness was associated with better diets, hygiene, pre-natal care, and so on, tending independently to lower mortality rates. Such a tendency would be reflected in a misleading way in the

coefficient of per cent of births in hospitals. It was thought that this might also be reflected in higher hospital accreditations, due for example to greater public pressure on hospitals to achieve accreditation; if so the inclusion of this factor would tend to remove the misleading element if any from the coefficient of X_1. However, the accreditations factor was not significant, and had little effect on the coefficient of X_1. All that this proves, of course, is that the use of this variable was not fruitful. Regressions were also calculated using assets per hospital as an additional variable, and this factor also proved insignificant.

Regression 12 may be compared with the basic regression 2. Concerning the form of the regression, particularly of the relationship between the dependent variable and the main independent variable X_1, this comparison makes it clear that the linear form is superior to the logarithmic. Regression 2, with an R^2 of .879, leaves only 12.1% of the variation in maternal death rates unexplained; regression 12, with an R^2 of .765, leaves 23.5%, or nearly twice as much, unexplained. The same point is established by the ratio of the coefficient of X_1 to its standard error; this ratio is over $6\frac{1}{2}$ in regression 2, but is less than $4\frac{1}{2}$ in regression 12. On this latter measure we may also compare the fit, or appropriateness of form, for the other two variables. X_2 fits almost equally well in both regressions, as the ratio of the coefficient to its standard error is just over 3 in both cases. (On analytical grounds one would lean toward the linear form in this case.) For X_3, regression 2 gives the better fit, although still not good enough to be conclusive.

Concerning the tendency of one variable to reflect the influence of another if the latter is omitted from the regression, it has already been remarked how the inclusion of X_2 (regressions 8, 9, 10 and 12) sharply reduces the apparent effect of X_3 (compare regressions 6 and 11). It is also worth noting that the regressions which include X_2 all have smaller coefficients (in absolute terms) for X_1 than the regressions without X_2, although here the difference is proportionately much less. Similarly it may be inferred that any other variables that have been omitted completely are no doubt partly (and misleadingly) reflected in the coefficient of X_1, but that the resulting error in the estimate of the effect of X_1 is almost certainly very small unless any of the omitted variables would make a large contribution, comparable to that of X_1, to the explanation of the variation in maternal death rates.

The regressions shown in table 4 represent the logarithmic form of regressions 5a, 5b, and 5c. Here there are two reasons for confirming the clear superiority of the linear form. First, the explanatory power is

much higher, as measured by the R^2, particularly when all states are included in the same regression (regression 5a as against 13a). Second, in both sets of regressions the coefficient of the change in X_1 is much larger (in absolute terms) for the low mortality states, in which this change was generally small, than for the high mortality states, in which this change was generally large. This would indicate that the best fit would have been obtained by a curve bending in the reverse direction from a logarithmic curve, showing the largest absolute effects on mortality in states with the lowest mortality rates. Because of the absence of other important variables from these regressions, however, this point was not worth pursuing.

ADAPTATIONS IN THE PHYSIQUE OF AMERICAN ABORIGINES TO NUTRITIONAL FACTORS

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INTRODUCTION

A strong inverse correlation has been shown to exist between the mean body weights of many human populations and the temperatures of the places where they reside (Roberts 1953). Yet there are factors other than environmental temperatures that influence body weights. The figures assembled by Roberts (1953, pp. 542-544) on 116 native samples around the world indicate that only 36% of the total variance in mean body weights can be attributed to mean annual temperatures of their locations. About 30% more of this variance is accounted for when mean statures are added to the weight-temperature relationship, while the remainder of about 33% is attributable to other factors. Examination of the possible nature of these factors is the principal purpose of the present paper, which is also an extension of that portion of Roberts' (1953) study dealing with the aboriginal populations of the New World. For this purpose these populations are nearly ideal, since the Americas present a simpler pattern of human settlement than any other continental area, having undergone a relatively late occupation through a single port of entry at Bering Strait.

MATERIALS AND METHODS

Sixty adult male Eskimo and American Indian samples—almost four times as many as Roberts studied—were assembled from published and unpublished sources (see table 1). Insofar as possible all mean weights are without clothing; the footnotes to table 1 indicate where adjustments were made to allow for clothing in the published figures. Where possible, samples were reseriated to eliminate teen-agers suspected of not having achieved full adult weight and stature. Mean ages which range from 28 to 45 years are available for about half the 60 samples, but appreciable weight increases with age can be anticipated only when strong acculturation is present. People living close to the land and practicing a basically

TABLE 1

TABLE 1

Weight, Stature, and Environmental Temperature Relationships in Adult Male American Aborigines

SERIES CODE	SAMPLE	NO.	MEAN AGE	MEAN STATURE (CM)	MEAN WEIGHT (KG)	MEAN C. M. TEMP. (°F)	OBS. VS. EXP. WEIGHT WITH TEMP. (KG)*	OBS. VS. EXP. WEIGHT W. STAT. & TEMP. (KG)*	STATURE $\sqrt[3]{\text{WEIGHT}}$
	Eskimo								
1	Copper-Aivilik [1]	20	—	163.5	74.0n [1]	—27	3.2	*6.2*	38.94
2	Southwest Greenland	43	30-40	157.7	*64.3*	16	—1.0	*5.4*	39.36
3	Aleut	28	40.8	161.2	65.3	31	*4.7*	*5.3*	40.03
4	E. Greenland-Angmagsallik	162	—	162.0	64.1	16	0.8	1.5	40.48
5	S. W. Greenland-Julianhaab	121	—	159.3	61.1n [2]	21	—1.3	1.3	40.44
6	Point Barrow	51	35.2	161.3	66.0n [3]	—18	—2.2	0.9	39.91
7	E. Greenland-Angmagsallik	113/20	—	162.4	63.6	16	0.3	0.7	40.68
8	W. Hudson Bay-Chesterfield Inlet	30	—	162.7	65.3	—26	*—5.3*	—1.7	40.40
9	Miscellaneous Alaska	17	28.6	165.7	63.3	—9	—4.3	*—4.7*	41.58
10	St. Lawrence Island	27	27.9	165.0	63.2	3	—2.3	*—3.4*	41.42
	North American Indian								
11	Papago	219	—	168.8	71.4n	53	*14.6*	*6.4*	40.69
12	Iroquois-Seneca	507	30.7	173.3	72.8n	25	*11.1*	1.4	41.51
13	Navaho, Ganado-Piñon	166	c. 36	169.8	68.7n [4]	33	*8.4*	1.2	41.46
14	Hopi—mostly Oraibi	276	—	161.1	60.8	33	0.5	0.6	40.97

TABLE 1 (Continued)

SERIES CODE	SAMPLE	NO.	MEAN AGE	MEAN STATURE (CM)	MEAN WEIGHT (KG)	MEAN C. M. TEMP. (°F)	OBS. VS. EXP. WEIGHT WITH TEMP. (KG)	OBS. VS. EXP. WEIGHT W. STAT. & TEMP. (KG)	STATURE / $\sqrt[3]{\text{WEIGHT}}$
15	Choctaw	26	32.2	171.4	66.1 [5]	49	*8.6*	—1.5	42.39
16	Zuni	348	—	161.4	56.3	35	—*3.6*	—*4.0*	42.11
17	Navaho	125	—	169.6	62.7	33	2.4	—*5.8*	42.69
	Middle American Indian								
18	Maya-Tzeltal (Huixtán)	50	—	153.6	53.9n [6]	55	—2.5	*3.2*	40.68
19	Maya-Yucatan	120	—	155.4	54.2n	72	0.8	2.5	41.04
20	Maya-Yucatan	69	30.6	155.1	53.5n	72	0.1	2.1	41.16
21	Maya-Chol (Tumbalá)	100	c. 33	158.6	57.4n [7]	62	2.2	2.0	41.11
22	Maya-Tojolabales (Independencia)	100	—	158.6	56.6	61	1.2	1.2	41.28
23	Maya-Yucatan	163	—	154.7	52.9	72	—0.5	1.8	41.21
24	Tarascan—Janitzio	116	c. 34	160.0	58.1	58	2.2	1.2	41.31
25	Maya-Quiché	30	young	159.3	57.6	55	1.2	1.0	41.25
26	Tarahumare-Norogachic	50	—	163.0	60.0n	64	*5.2*	1.0	41.64
27	Maya-Tzeltal (Amatenango)	50	—	158.2	56.0n [6]	61	0.6	0.9	41.35
28	Mixtec-Tilantongo	148	—	155.9	54.0	59	—1.7	0.7	41.24
29	Maya-Tzotzil (Chamula)	100	young	155.7	53.3n [6]	55	—*3.1*	—0.2	41.37
30	Maya-Kanhobal (Soloma)	74	—	154.8	52.6n	54	—4.0	—0.2	41.39
31	Yaqui	100	—	166.7	62.0n	62	*6.8*	—0.3	42.11
32	Otomi-El Zapote	100	35.1	157.0	52.6n	67	—1.7	—0.9	41.97
33	Chicantec	45	—	157.6	52.8	71	—0.8	—0.9	42.01

TABLE 1 (Continued)

33	Chicantec	45	—	157.6	52.8	71	—0.8	—0.9	42.01

TABLE 1 (Continued)

SERIES CODE	SAMPLE	NO.	MEAN AGE	MEAN STATURE (CM)	MEAN WEIGHT (KG)	MEAN C. M. TEMP. (°F)	OBS. VS. EXP. WEIGHT WITH TEMP. (KG)	OBS. VS. EXP. WEIGHT W. STAT. & TEMP. (KG)	STATURE / $\sqrt[3]{\text{WEIGHT}}$
34	Nahua-Huatusco	99	36.9	158.6	54.7n	58	—1.2	—1.0	41.78
35	Maya-Tzotzil (Zinacantan)	25	—	158.4	54.7n [6]	55	—1.8	—1.1	41.73
36	Nahua-Pajapan	100	34.2	161.9	56.4n	70	2.6	—1.1	42.23
37	Maya-Quiché (Sta Clara)	63	33.6	154.2	51.1n	54	*—5.5*	—1.2	41.55
38	Tepehua-Pisaflores	100	32.2	157.7	52.8n	67	—1.5	—1.3	42.04
39	Mixtec—nr. Oaxaca	78	—	154.2	50.2n [6]	61	*—5.2*	—1.5	41.80
40	Otomi-Ixmilquilpan	105	28.2	157.6	53.2n	58	—2.7	—1.7	41.90
41	Maya-Tzeltal	47	—	155.6	51.4n [6]	58	—4.4	—1.8	41.85
42	Maya-Cakchiquel (Patzún)	72	—	155.3	51.4n	54	*—5.2*	—1.9	41.77
43	Maya-Cakchiquel (Soloma)	82	—	154.8	50.8n	54	*—5.8*	—2.0	41.80
44	Tarascan-Paracho	47	c. 34	161.4	56.1	57	0.1	—2.1	42.16
45	Totonac-Papantla	100	33.2	158.0	52.0n	70	—1.8	—2.1	42.33
46	Zapotec—near Oaxaca	50	—	155.4	50.3n [6]	61	*—5.3*	—2.4	42.12
47	Trique-San Andres	101	20-40	156.5	50.8	63	—4.2	—2.7	42.26
48	Nahua-Zongolica	100	39.2	154.8	49.5n	60	*—6.0*	—2.8	42.16
49	Nahua-Chiconamel	99	32.9	157.0	50.3n	68	—3.8	*—3.1*	42.56
50	Huastec-Tantoyuca	100	32.8	157.2	50.4n	68	—3.7	*—3.2*	42.56
51	Nahua-Tepoztlán	159	33.0	163.1	56.4	58	0.5	*—3.2*	42.54
52	Nahua-Gabriel Mariaca	36	44.9	163.4	56.6	58	0.7	*—3.3*	42.56
53	Nahua-Tlahuica (Otepec)	50	—	160.0	52.0	59	—3.7	*—4.8*	42.87

TABLE 1 (Continued)

SERIES CODE	SAMPLE	NO.	MEAN AGE	MEAN STATURE (CM)	MEAN WEIGHT (KG)	MEAN C. M. TEMP. (°F)	OBS. VS. EXP. WEIGHT WITH TEMP. (KG)	OBS. VS. EXP. WEIGHT W. STAT. & TEMP. (KG)	STATURE / $\sqrt[3]{\text{WEIGHT}}$
	South American Indian								
54	Araucanian-Mapuche	31	—	163.3	66.8	59	*11.1*	*7.1*	40.25
55	Tucano-Decana	10	—	154.2	55.8n	78	3.4	*5.7*	40.35
56	Oajana	15	—	155.0	55.1	79	2.9	*4.4*	40.73
57	Trumai	11	—	158.8	58.4n	74	*5.3*	*4.0*	40.93
58	Wai Wai	20	30.1	158.6	56.6n	80	*4.6*	2.9	41.31
59	Quechua-Morococha	478	20-34	159.0	55.4n	40	—3.6	—2.4	41.71
60	Quechua-Vicos	180	32.0	154.6	51.5n	50	*—5.8*	—1.5	41.55

* Differences $> 1\sigma$ are italicized.

n Reduced to nude weight, either specified by author or deductions made here.

[1] A pooled sample composed of Jenness' 11 Copper Eskimo and Boas' 9 Aivilik-Kinipetu. On the assumption that Boas' published weight included clothing, 3.6 kg (8 lb.) were deducted here.

[2] Fabricius-Hansen weighed his subjects fully dressed but without footgear in the summer time; hence 1 kg was deducted from his published mean.

[3] On the assumption that Ray did not deduct for clothing, 3.6 kg were deducted from the published mean.

[4] Two kilograms deducted from published mean.

[5] Collins' (1925, p. 435) stated that the mean weight for 35 Choctaw men was 149.2 lb (67.8 kg), but this included seven men whose weights were only estimated. Removal of these seven men from the sample reduces the mean weight to 66.1 kg.

[6] Leche (personal communication) states that all her samples were weighed without footgear but in two light garments amounting to about 0.5 kg. This amount is deducted here.

[7] One kilogram deducted for clothing.

References to Table 1

[7] One kilogram deducted for clothing.

References to Table 1

SERIES CODE	AUTHOR	SERIES CODE	AUTHOR	SERIES CODE	AUTHOR
1	Jenness 1923; Boas 1901	21	Gould, H. N. 1946	41	Leche *et al.* 1944
2	Luther, n. d.	22	Basauri, C. 1931	42	Stewart, n. d.
3	Laughlin 1951	23	Williams 1931	43	Stewart, n. d.
4	Skeller 1954	24	Gomez Robleda 1943	44	Gomez Robleda 1943
5	Fabricius-Hansen 1949	25	Crile & Quiring 1939b	45	Faulhaber 1950-1956
6	Ray 1885	26	Basauri, C. 1929	46	Leche 1936a
7	Gessian 1958	27	Leche *et al.* 1944	47	Comas 1944
8	Crile & Quiring 1939a	28	Romero, n. d.	48	Faulhaber 1950-1956
9	Rodahl & Edwards 1952	29	Leche 1936a	49	Faulhaber 1950-1956
10	Bullerud *et al.* 1950	30	Stewart, n. d.	50	Faulhaber 1950-1956
11	Gabel 1949	31	Seltzer 1936	51	Field 1954
12	Gould, B. A. 1869	32	Faulhaber 1950-1956	52	Field 1954
13	Darby *et al.* 1956	33	D'Aloja 1939	53	Basauri, M., n. d.
14	Gabel 1949	34	Faulhaber 1950-1956	54	Pi-Suñer 1933
15	Collins 1925	35	Leche *et al.* 1944	55	Bastos D'Avila 1950
16	Gabel 1949	36	Faulhaber 1950-1956	56	Droogleever-Fortuyn 1946
17	Gabel 1949	37	Stewart, n. d.	57	Ranke 1910
18	Leche *et al.* 1944	38	Faulhaber 1950-1956	58	Newman, M. T., & Jones, n. d.
19	Steggerda 1941	39	Leche 1936b	59	Hurtado 1932
20	Steggerda 1932	40	Schreider 1954	60	Newman, M. T., *et al.*, n. d.

aboriginal food economy may not show weight increases past the mid-thirties.

The geographic distribution of the 60 New World samples is regrettably uneven: 36 are from Middle America, 10 from the Eskimo area, and seven each from the rest of North America and from South America. Achievement of even this distribution necessitated use of nine samples represented by less than 30 individuals each. Yet five of these samples (table 1, series code 3, 15, 56, 57, 58) are drawn from remnant populations and hence are more representative of them than otherwise would be the case. No sample with strong racial admixture was used in table 1, although the majority may contain individuals of partially non-aboriginal ancestry.

Roberts (1953) used mean annual temperatures as a measure of climate, but it has since been shown (R. W. Newman and Munro 1955, p. 8) that mean coldest month temperatures correlate more highly with body weights and hence are used in the present study. Within the United States the mean coldest month temperatures used in table 1 were interpolated from station data taken from the U. S. Weather Bureau's (1954) report for January. Outside the U. S. practically all the temperatures are interpolations from one of the Environmental Protection Research Division's Technical Reports (1958a and b, 1959). The American Geographic Society's Millionth Map Series was consistently used in making the interpolations.

The statistical methods employed in this study are quite standard and need no explanation. The zero order correlation coefficients were calculated with a system of arithmetic checks (Ezekiel 1941, pp. 245-258), and the multiple correlation was done with the Doolittle Method with check sum (Ezekiel 1941, pp. 261-268).

ANALYSIS

I. *Weight-Temperature Relationships*

The correlation coefficient r of -0.493 between mean weights and mean annual temperatures reported by Roberts (1953, p. 549) for 16 New World samples is of doubtful significance ($P = .10$) because of only 13 degrees of freedom. With 53 of the present samples (including Roberts' 16), r is -0.668, highly significant at the .001 level, when mean annual temperatures are used. When mean coldest month temperatures are substituted for the mean annual figures, the r for 60 samples becomes -0.729, significant at the .0001 level. This higher correlation between

TABLE 2

Measures of Relationships in Body Weight, Stature and Environmental Temperature

MEASURES OF RELATIONSHIP	PRESENT WORLD SERIES SERIES (60)		ROBERTS' NEW WORLD SERIES (16)		ROBERTS' WORLD SERIES (116)		NEWMAN AND MUNRO'S U. S. WHITES (15,216)	
	r	Signif. at	*r*	Signif. at	*r*	Signif. at	*r*	Signif. at
Zero order correlations								
Weight-Stature	+.805	.0001	+.818	.001	+.734	.001	+.655	.01
Weight-Mean annual temp.	—		—.493	.10	—.600	.001	—.460	.01
Weight-mean coldest month temp.	—.729	.0001	—	—	—		—.528	.01
Stature-mean annual temp.	—		—	—	.351	.001	—.044	Not
Stature-mean coldest month temp.	—.470	.0001	—	—	—		—.111	Not
Partial correlations								
Weight-stature (Holding temperature constant)	+.766	.0001	—	—	+.698 [1]	.001	—	—
Weight-temperature (Holding stature constant)	—.670	.0001	—	—	—.538 [1]	.001	—.483	.01
Stature-temperature (Holding weight constant)	+.289	.065	—	—	+.164 [1]	Not	—.187	Not

[1] Based on mean annual temperatures; all other partial correlations are based on mean coldest month temperatures.

mean weights and mean coldest month temperatures is in accord with R. W. Newman and Munro's (1955, p. 8) findings, although the difference between — 0.668 and — 0.729 is not significant ($P = .54$).

In all four studies shown in table 2, mean weights are more strongly correlated with stature than with temperature. This means that mean body weights can be somewhat more accurately predicted from statures than from temperatures. Mean statures are also inversely correlated with temperature: — 0.351 for Roberts' 116 world samples and — 0.470 for the 60 New World samples, significant at the .001 and .0001 levels, respectively. The influence of stature in the weight-temperature relationship can be tested by means of partial correlations (table 2). These agree with the findings of Roberts (1953, pp. 542-544) and R. W. Newman and Munro (1955, p. 9) on two principal points: 1. With stature held constant, the weight-temperature relationship remains statistically significant; 2. with weight constant, the stature-temperature correlation becomes not significant and even ceases to be inverse. These partial correlations clearly indicate that the basic relationship is between the body weight of a population and its environmental temperature. Only because of its strong positive correlation to weight does stature show a significant correlation with temperature. With body weight the basic ecological correlate, it can be said with Roberts (1953, p. 549) that stature is responsible only for additional variation in weight. Accordingly, a better description of the variation of body weight is to be seen in multiple correlation of weight with temperature *and* stature (see II).

Roberts' (1953, p. 548) regression equation for weight on temperature in 16 New World samples is: Weight = 66.2 — .123 (Temperature). The same equation, using mean coldest month temperatures for the 60 New World samples, is: Weight = 66.07 — .176 (Temperature). This regression line is shown graphically in figure 1. Much of the increased slope of the regression line—from .123 to .176 kg mean weight increase for each 1° F drop in mean temperature—may be attributed to the use of mean coldest month rather than mean annual temperatures. On a world basis, the .176 kg increase per 1° F decrease is rather moderate. Roberts' (1953, Fig. 8) 28 African and 20 European samples show .512 and .414 kg. increases, respectively. Only his 29 East Mongoloid samples have a lower increase of .100 kg, but this series did not include a number of notably heavy Siberian samples. Whether the racial-geographic differences in the slope of the weight-temperature regression are due to genetic or cultural factors, or indeed are an artefact of sampling, is a problem for future research.

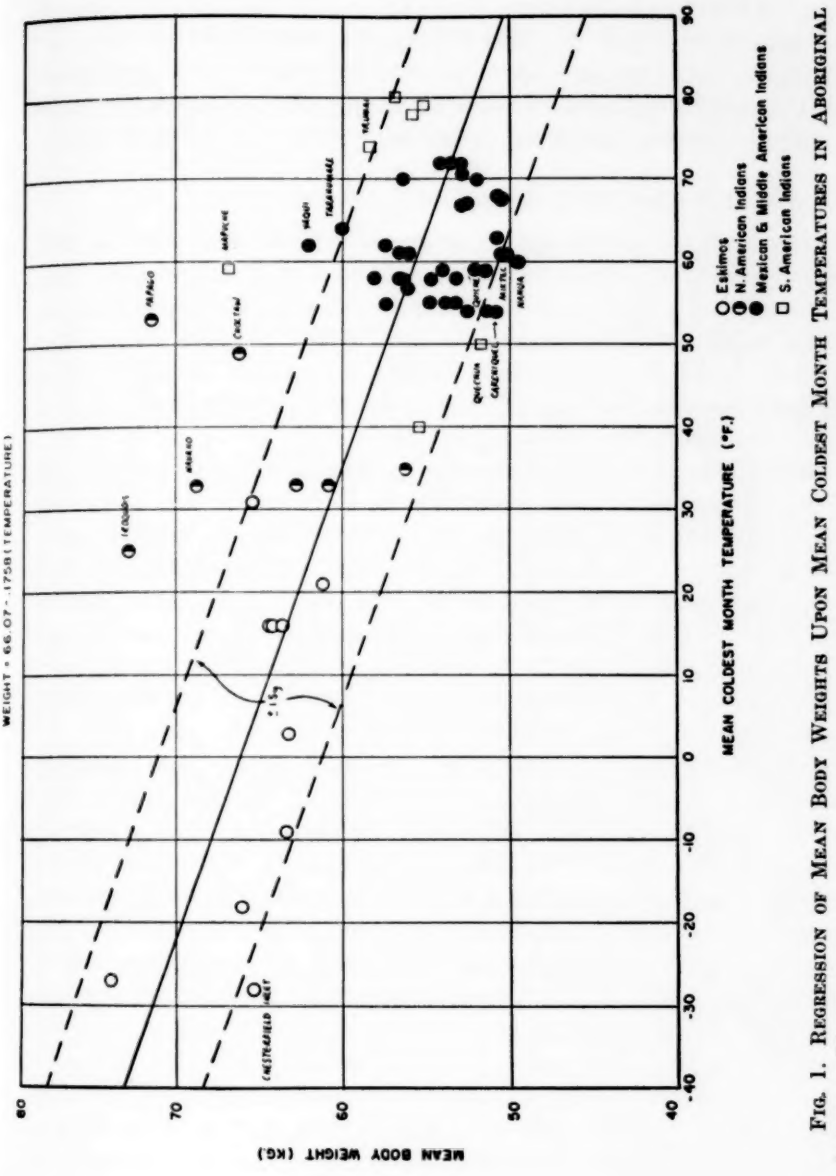

FIG. 1. REGRESSION OF MEAN BODY WEIGHTS UPON MEAN COLDEST MONTH TEMPERATURES IN ABORIGINAL AMERICAN MALES.

The regression equation for the 60 New World samples has a notably high σ of 4.59 kg (see figure 1). The r of -0.729 between mean weights and temperatures indicates that a little more than 50% of the total variance in mean weights can be attributed to environmental temperatures. Certainly one of the factors accounting for the residual variance is stature, which is considered in the multiple correlation that follows.

II. *Weight-Stature and Temperature Relationship*

The multiple correlation R of mean weights with statures and temperatures is very strong at 0.878 for the 60 New World samples. This R is somewhat greater than the 0.820 for Roberts' 116 world samples, largely because of better temperature data. With R at 0.878, almost 80% of the total variance in mean body weights is ascribable to mean statures and temperatures. The residual 20% of the mean weight variance, then, is due to other factors discussed in III.

Within the nearly 80% coverage by stature and temperature, the former has a slightly greater contribution as judged by the zero order and partial correlations of table 2. Beta coefficients, however, give stature a clear predominance: Weight — Stature·Temperature = 0.644; Weight-Temperature·Stature = 0.378. The predominance of the stature factor in predicting weight noted previously is readily understandable on anatomical grounds in that the total range of human body build has rather restricted normal mean limits. In the 60 New World samples this range exemplified by the stature/ $\sqrt[3]{\text{weight}}$ ratio (table 1) is especially restricted, as might be anticipated by the high r between weight and stature. This mean range of stature/ $\sqrt[3]{\text{weight}}$ is from 38.9 to 42.9, whereas the theoretical range for individual ratings extends from below 33 to over 47 (Hooton *et al.* 1951). In addition to demonstrating the low mean range in body build of the 60 New World samples, the stature/ $\sqrt[3]{\text{weight}}$ ratios closely correspond to the differences between observed weights and weights calculated from the multiple regression equation (see table 1). Thus those samples with $> 1\sigma$ greater observed than calculated weights have mean stature/$\sqrt[3]{\text{weight}}$ ratios of 39 — 41; those with $> 1\sigma$ greater calculated than observed weights have ratios of 41 — 43. This paralleling of weight deviations and stature/ $\sqrt[3]{\text{weight}}$ ratios indicates that the position of a population relative to the weight-stature and temperature regression plane serves as a good measure of its average *body proportions*. The position of a population in its mean *body mass* is best seen in relation to the simple weight-temperature regression.

The multiple regression equation for the 60 New World samples is Weight = .8584 (Stature) — .0927 (Temperature) — 75.03 with a σ of 2.92 kg. This σ is appreciably lower than the σ of 4.59 kg for the weight-temperature regression equation, and indicates better fit of the data to the multiple regression equation. The three-dimensional relationships expressed in the multiple regression equation are illustrated by the isometric plot shown in figure 2.

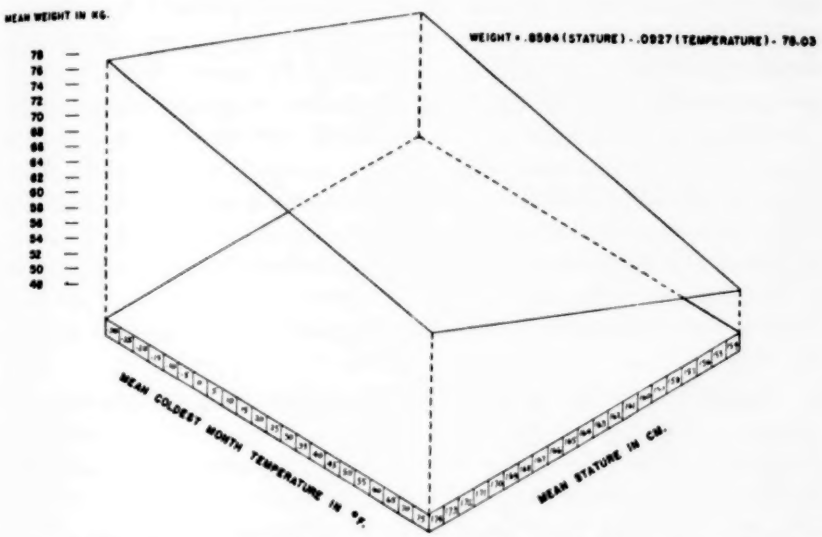

FIG. 2. REGRESSION OF MEAN BODY WEIGHTS ON MEAN STATURES AND MEAN COLDEST MONTH TEMPERATURES IN ABORIGINAL AMERICAN MALES.

III. *Variation of Body Mass and Proportions in the Individual New World Samples*

In appraising average body mass and proportions in the individual population samples, the immediate problem is to explain their observed deviations in body weight from the two regression equations. The most extreme deviations are seen in the 19 samples $> 1\sigma$ from the weight-temperature regression line (figure 1) and the 18 samples $> 1\sigma$ from the weight-stature and temperature regression plane. These and the lesser deviations in observed weight are best examined in the following groupings of individual samples.

A. *Eskimo*

Within the 10 Eskimo samples, the correlation coefficient r between mean weight and mean coldest month temperature is — 0.624, doubtfully significant at the .06 level because of only 7 degrees of freedom. More Eskimo samples would probably demonstrate a highly significant body mass increase from Low to High Arctic.

The most significant deviations from the weight-temperature regression line are the Aleut, who are heavy, and the Chesterfield Inlet Eskimo who are light in weight over the 1σ level. The Aleut sample has a high mean age of 40.8 years, which considering the degree of acculturation present, partly accounts for the greater mean weight. Yet the Aleuts are also a laterally built people, as judged from the 5.4 kg excess of observed versus calculated weight by the multiple regression equation. They are the only Eskimo sample that is both large in body mass and lateral in body proportions over the 1σ level. The low body weight of the Chesterfield Inlet sample relative to their environmental temperature is particularly enigmatic, since Boas' (1901) 9 Oivilik and Kinipetu Eskimo immediately to the north averaged 8 kg more (see table 1, footnote 1).

Weight deviations $> 1\sigma$ relative to stature and temperature are present in half the Eskimo samples. Excess of observed over calculated weight is present in the Aleut and the Copper-Aivilik and one Southwest Greenland Eskimo samples, and indicates especially lateral body build. Laughlin (1951, p. 104) noted this feature in the Aleut, and Jenness (1928, p. 38), did the same for the Copper Eskimo. Since Luther's (N. D.) race mixture study in Southwest Greenland sorted out the purest Eskimo (Series code 2) on a genealogical and phenotypic basis, stockiness and accompanying low stature may have been included in the sorting criteria. If this is true, it explains why one Southwest Greenland Eskimo (Series code 2) is more lateral in build than the other (Series code 5).

At the opposite end of the Eskimo scale of body build are the Miscellaneous Alaskan and St. Lawrence Island samples (Series code 9 and 10). Both show negative weight deviations in excess of 1σ, and in consequence are as linear in build as the average American Indian. It is also notable that these two Alaskan Eskimo samples are taller than most Eskimo. The linearity of the St. Lawrence Island Eskimo has a ready migrational explanation. They are not American Eskimo at all, but are directly derived from Asia, where they are closely related to the Chukchi—also

a taller and more linear people (H. B. Collins, Jr., personal communication).

Rodahl and Edwards (1952), were unaware of the unusual linearity of the Miscellaneous Alaskan and St. Lawrence Island samples, which they used to show that the DuBois height-weight formula for estimating skin surface area was applicable to Eskimo without significant error. This demonstration was that the skin area estimated by the height-weight formula only exceeded by 0.8 % the area calculated by the more accurate DuBois linear formula based on lengths and circumferences of the eight body segments. Yet, as we have seen, all Eskimo are not alike in body build. Most are more lateral in body build, probably show the Allen's Rule effect of longer trunks and shorter legs, and in consequence are likely to have appreciably lower skin surface areas as calculated by the linear method.

The variation in Eskimo body mass also led R. W. Newman and Munro (1955, p. 13) to develop figures grossly underestimating their weight relative to environmental temperature. Pooling the three Eskimo samples cited by Roberts (1953, App. A; Series code 5, 8, and 9-10 in table 1 here), they calculated a mean body weight of 62.9 kg for 190 individuals, with a weighted mean annual temperature of 29.2° F. At this temperature, mean weight calculated by Roberts' New World regression equation is 66.7 kg, 3.8 kg higher than the observed mean. On this basis R. W. Newman and Munro claimed that the Eskimo would fit the regression equation better at a mean annual temperature of over 40° F, like that of Maine or Minnesota. Their reasoning behind this claim was that because of superb cultural adjustment the Eskimo were insufficiently cold-stressed to conform to the weight-temperature regression.

This argument is subject to two principal emendations. First, the pooled sample of 190 Eskimo is heavily overloaded by the 121 South Greenlanders from the far southerly Julianhaab District. At almost the 50° N. Latitude of Cape Farewell, these people not only live at the southernmost edge of the Low Arctic, but are also acculturated in their diet. The Chesterfield Inlet sample, as we have seen previously, is the only one with a mean weight deficiency $> 1\sigma$, and the combined St. Lawrence Island-Miscellaneous Alaskan sample is notably linear in body proportions. In short, R. W. Newman and Munro's pooled Eskimo sample distinctly favors the lighter in weight and more linear segment of the range in these Arctic people. Secondly, the same procedure applied to the 10 Eskimo samples (519 individuals) yields weighted means of

64.03 kg for weight and 9° F for coldest month temperature. Calculated weight at 9° F by the new regression equation given above is 64.48 kg only .45 kg more than the observed. This close approximation of observed and calculated weights makes it clear that on the average the Eskimo conform to the New World weight-temperature regression equation developed in this paper.

B. *North American Indians*

The Papago, Iroquois, Choctaw and the Navaho sample from Ganado and Piñon show mean weight-temperature excesses around $+ 2\sigma$. These high mean weights are due principally to tall stature, since the excesses largely disappear when stature is brought into the weight-temperature relationship. An exception is the Papago sample, which is not only heavy in body mass but lateral in proportions, as the excess of 6.4 kg in weight relative to stature and temperature indicates. Gabel (1949, p. 16) suggests that the great body weight of the Papago is due to "... an inherent tendency toward obesity." But it is more than that. Age for age the 115 Papago boys and girls studied by Vavich *et al.* (1954, p. 124) average at least 10 kg heavier than Martin's (1953, p. 17) pooled U. S. White series showing closely comparable statures. This 10 kg difference can hardly be attributed to body fat, and clearly marks the Papago as a laterally built people. That they may be somewhat obese as well is suggested by their high carbohydrate diet (Vavich *et al.* 1954, p. 123), and by the employment of Gabel's sample in the Indian Division of the Civilian Conservation Corps at the time he made his study. This employment undoubtedly brought a substantial dietary supplement to these Papago men.

The Iroquois of a century ago, the Choctaw of 35 years ago in Mississippi, and the modern Navaho from Ganado and Piñon do not express any unusual lateralness of build. Indeed, the Choctaw show a 1.5 kg deficiency in weight relative to stature and temperature, and are rather more linear than one would expect of Eastern Indians. The most likely explanations lie in their workload as farmers and some Anglo-Saxon White and Negro admixture. What little is known of Iroquois diet 100 years ago suggests high carbohydrate and fat intake (M. T. Newman 1957, pp. 358-359), but they are only 1.4 kg over the regression plane for their stature and temperature. Their workload may have been great enough to keep down excess accumulations of body fat, as seems to be the case for the Navaho as well. A real reduction of workload coupled with dietary changes attendant upon reservation life can result

in pronounced obesity, as Eggan and Pijoan (1943, pp. 15-16) have shown for the Nevada Shoshone.

It may be that a high workload and a temporary dietary insufficiency can account for the 6 kg mean weight difference between the Ganado and Piñon Navaho studied in the early 1950s and the Navaho studied 20 years earlier by Seltzer (Series code 17). These two samples come from the same northeastern corner of Arizona, and are not likely to represent a regional difference. Age could only be a factor if Seltzer's sample were overloaded with teen-agers and old men. Nothing other than generalities of diet are available for the early 1930s, but in the early 1950s the nutritional status of the Navaho at Ganado and Piñon was reasonably good in a caloric sense, with no problems of anemia, rickets, or protein deficiency, and only a somewhat low vitamin C intake (Darby *et al.* 1956, p.75). Thus for lack of a better explanation it may be that the lower body weight without a diminution of stature in Seltzer's Navaho may represent a temporary dietary linearity. This Navaho sample and the one from Zuni (Series code 16) show deficiencies of observed weight relative to stature and temperature of 5.8 and 4.0 kg, respectively. They are the most linear of the North American Indians figured in table 1.

C. *Middle American Indians*

The only Middle American Indians with observed mean weights $> +1\sigma$ than those calculated by the weight-temperature regression equation are the taller samples from Northern Mexico—the Yaqui of Sonora and the Tarahumare of Chihuahua. When stature is brought into the relationship, both samples are quite average in body proportions, with the Yaqui somewhat the more linear of the two. The rest of the Middle American samples listed in table 1 are from the Central and Southern sections of Mexico and from Guatemala. For the most part they are short-statured (< 160 cm) people and their mean weights are commensurately low. Figure 1 clearly shows that none of these 34 samples have observed weights $> 1\sigma$ above the regression line; indeed, most are below it. Their average weight deficiency of 1.9 kg suggests that some unaccounted for factor operates to depress body mass in this region.

Six of the 34 samples have mean weight deficiencies $> 1\sigma$ and are all highland people living between 1,300 and 2,200 meters elevation. These are the three Lake Atitlán Basin samples, the Mixtec and Zapotec near Oaxaca, and the Nahua from Zongolica in Vera Cruz (Series code 37, 39, 42, 43, 46, 48). While the lower environmental temperatures consistent with altitude result in greater weights as calculated by the regres-

sion equation, observed weights average only 0.6 kg higher in the 23 highland than in the 11 lowland samples. In consequence, the discrepancies between observed and calculated weights bulk larger in highland people. This suggests that life in the Middle American highlands involves extra stressing over and above the chilly environmental temperatures.

The most likely hypothesis to explain this additional stressing may well lie in the area of food intake and energy output. The diets of Middle American Indians have often been claimed to be qualitatively and quantitatively deficient (cf. Comas 1943, Fuente 1945), and the few studies cited in table 3 show a clearly hypocaloric trend. These mean daily per capita intakes represent only rough estimates of the food put to metabolic use, and there are perhaps as many unappraised factors favoring overestimation as there are favoring underestimation of caloric intake and utilization. There is no reason to believe that these factors cancel each other. The recommended levels of caloric intake in table 3 are also rough estimates, and are based upon the unvalidated concept that after adjustments are made for sex, age, body weight, workload, and environmental temperature, caloric requirements are about the same the world over.

Table 3 shows that the mean caloric intakes of the six samples closely straddle the recommended levels applied to them. These caloric intakes are probably adequate to sustain small-bodied people, although in some cases barely so. But could this level of caloric intake support larger-bodied people, particularly during phases of growth? Or is the small body mass so characteristic of the Middle American highlands partly a product of the quite limited calories available per capita? Upland fields of mediocre quality have often been relegated to Indian communities, with the better lands held by Ladinos. Mediocre land, inefficient agricultural techniques, and unimproved seed and stock coupled with rapid population increases inevitably limit the per capita food intake.

In addition to the caloric limitations, broad areas of Middle America are noted for low protein intake. As Scrimshaw et al. (1955b) have shown, severe protein deficiencies (kwashiorkor) are most prevalent in preschool children of poorer Guatemalan families and has a heavily retarding influence on their growth and maturation—indeed, if they live at all. Other commonly reported deficiencies are in vitamins A, the B-complex, and C. To these low nutritional ceilings may be added the omnipresence of intestinal parasites, some of which constitute a very appreciable nutritional drain on their hosts if the infestation is massive. In two studied communities, the incidence of *Ascaris lumbricoides,* one

TABLE 3

Mean Daily Caloric Intake per Capita in Middle American Indians

GROUP	LOCALITY	AUTHOR	ELEVATION (METERS)	MEAN DAILY CALORIC INTAKE	PERCENT OF RECOMMENDATIONS
Otomi	Mezquital Valley	Anderson *et al.* 1946	1.650	1706	70
Tarascan	Cherán, Michoacan	Beals & Hatcher 1943	2.160	2501	75
	Guatemala				
Maya-Cakchiquel	Magdalena Milpas Altas	Flores & Reh 1955a	1.718	2013	115
" "	Santo Domingo Xenacoj	" " " 1955b	1.720	2283	114
" "	San Antonio Aguas Caliente	" " " 1955c	1.445	2170	105
" "	Sta. Maria Cauqué	" " " 1955d	1.979	2290	118

of the most debilitating of the roundworms, is 80% and over in children from Magdalena Milpas Altas and Santo Domingo Xenacoj, Guatemala (Flores and Reh 1955a and b). Furthermore, there is evidence of a not wholly clear nature that when childhood infections are controlled by antibiotics, growth proceeds at a faster pace in Guatemalan school populations (Scrimshaw *et al.* 1955a).

The workload or energy output of Middle American Indians may very well be greater than allowed for in the " reference man " used in INCAP's (1953) standards. This " reference man " works eight hours a day and walks 5-10 km on level ground. The revised recommendations for 1954 (INCAP 1955) recognize the greater energy output of Middle American subsistence farmers and add 200 calories a day to their requirement. But the earlier recommendations do not take this into account. Nor do any of the recommendations allow for the full loss of calories in keeping warm during nights approaching freezing temperatures in the highlands. Utilization of extra calories for body-warming may account for the elevated basal metabolic rates reported in winter months for both Highland and Lowland Maya (Roberts and Newman, n. d.).

Taken together, the rather low nutritional ceiling, unfavorable disease environment, heavy workload, and some cold-stressing may leave rather little left over for the processes of growth and maturation. Small adult body mass appears to be the result. With the assurance of the archeological record that there has been sufficient time for selection to operate, it is probable that the slow growth and small body size constitute long-term adaptations to a difficult total environment. Conceivably this adaptive process was hastened by the per capita nutritional pinch that may have been occasioned by rapid population expansion.

When stature is brought into the weight-temperature relationship, 25 of the 36 Middle American samples fall below the multiple regression plane. The mean deficiency in weight relative to stature and temperature is 0.8 kg. Only one sample shows a mean observed weight $> 1\sigma$ greater than its calculated weight. This is a Tzeltal-speaking sample from Huixtán in Highland Chiapas—the most short-statured of all 15 Maya populations sampled. In this case the shorter stature is a large part of the explanation for the unusually lateral body proportions.

Five other Middle American samples—four of them Nahua-speaking —are deficient in mean weight relative to stature and temperature by over 1σ. These five samples come from both highland and lowland Central Mexico: Nahua from Chiconamel and Huastec from Tantoyuca in the Vera Cruz lowlands; and Nahua from Tepoztlán, Gabriel Mariaca

and Otepec in upland Morelos (Series code 49-53). Three other Nahua samples from lowland Pajapan and highland Huatusco and Zongolica in Vera Cruz (Series code 34, 36, 48) are also on the linear side of the regression plane. All seven Nahua samples have a mean weight-stature and temperature deficiency of 2.7 kg, which stands in decided contrast to the + 0.7 kg mean for 15 Maya samples. In the stature/ $\sqrt[3]{\text{weight}}$ ratio, which is independent of temperature, the Nahua average 42.38 and the Maya 41.37. One ratio unit is a considerable difference in the light of the four unit mean range for the entire New World. These data clearly indicate an especially strong tendency toward linearity in Nahua-speaking Indians. It is most unlikely that all seven Nahua populations from both highlands and lowlands were studied during a period of temporary agricultural adversity and very short food supply. Thus the explanation can hardly be dietary linearity, but rather may be a strong genetic tendency toward linearity in these Nahua people.

D. *South American Indians*

The first five South American Indian samples listed in table 1 are from lowland areas, and all have high mean body weights and rather lateral body builds. All but the Araucanian Mapuche are tropical forest people living on a full and variegated dietary that is rich in fish and game (Levi-Strauss 1948, pp. 324-326; M. T. Newman and Jones, n. d.). In the case of the Wai Wai (Series code 58), clinical examinations revealed hardly a sign of nutritional deficiencies, although the population was plagued by malaria and other parasitic infections (M. T. Newman and Jones, n. d.).

For the two highland samples, the one from Morococha consists of Quechua mine workers operating at over 4,000 meters altitude. Their deficiencies in body mass and proportions are $< 1\sigma$, and are minor enough to be explained by Monge's (1953, p. 138) hypothesis that high-altitude people are physically comparable to trained athletes. The other highland sample from Vicos in the Callejon de Huaylas of Peru shows a mean weight-temperature deficiency of 5.8 kg, but in body proportions only averages 1.5 kg below the weight-stature and temperature plane. Thus they are small in body mass even as compared to other Quechua Indians, but are not particularly linear in build.

The most likely explanation for the low body mass in the Vicos Quechua lies in the same food-intake energy-output argument used for the Middle American highland people. Mean daily food intake from two dietary surveys (Collazos *et al.* 1954, p. 1228) averaged about 1500

calories per person, 70% of adjusted INCAP (1953) recommendations. Protein, calcium, vitamin A, and riboflavin intakes were, respectively, 60-76, 13-14, 2-3, and 46-66% of recommended levels. Other intakes were at acceptable levels. Both surveys were conducted in good crop years, and both missed the lean December-January season. Extra nutritional drain must have been occasioned by wide-spread and often severe parasite infestations (Payne et al. 1956), by cold-stressing through sleeping under light covers on open porches at night, and by a very heavy workload. These factors appear to be reflected in slow bodily growth and maturation (M. T. Newman and Collazos 1957), low bone mineralization until at least 17 years of age (Schraer and Newman 1958), and small adult body mass. Stated the other way around, this pattern of slow growth and small body mass would appear to be an advantageous adaptation to a difficult and at times hostile total environment.

CONCLUSIONS

The inverse relationship between mean body weights and mean environmental temperatures in native peoples of the Americas has been established on a statistically significant basis by the present study. The coefficient of correlation r in 60 Eskimo and Indian samples is -0.729, highly significant at the .0001 level. The regression equation for mean weights on mean coldest month temperatures is: Weight $= 66.07 - .176$ (Temperature), with a σ of 4.59 kg. Thus about 50% of the total variance in mean body weight of New World aborigines is attributable to environmental temperatures. When mean statures are added to the weight-temperature relationship, the multiple correlation of R is higher at 0.878, and the σ of the regression equation is reduced to 2.92 kg. The equation is: Weight $= .8584$ (Stature) $- .0927$ (Temperature) $- 75.03$. From the R it is apparent that almost 80% of the total variance in mean weight is due to stature and temperature. Conversely over 20% of the variance must be attributed to other factors, which are the primary subject of investigation here. These factors are best seen in analysis of the individual population samples.

Mean weight deviations of individual samples from the weight-temperature regression line (figure 1) reflect the excesses or deficiencies in body mass. Of the 10 samples with mean weight excesses $> 1\sigma$, five from the United States and Northern Mexico are heavy because they are tall. The remaining five heavy samples represent a miscellany of laterally-built peoples—the Aleut, Papago, and three South American lowland

groups. Tentatively their heaviness and lateral builds find primary explanation in heredity, although several of them may also reflect the obesity of acculturation in diet and a reduced workload. Seven of the eight samples deficient in mean weight by $> 1\sigma$ are from the highlands of Middle and South America, where a low nutritional ceiling, difficult disease environment, and heightened workload combine to make slow growth and maturation and small adult body mass an advantageously adaptive pattern.

The mean weight deviations from the weight-stature and temperature regression plan (figure 2) provide a measure of body proportions in general terms of linearity and laterality. The 10 samples with weight excesses $> 1\sigma$ in this respect have principal geographic foci in the Eskimo and the lowland South American areas. The lateral body builds of the Aleut, Copper-Aivilik, and Southwest Greenland samples may be explained at least in part by the Allen's rule phenomenon (see M. T. Newman 1953, p. 312) of long trunks and short legs—presumably a long-term cold-stress adaptation and hence conditioned by environmental temperatures. For lowland South America, four of the five laterally-built samples are from the tropical forest regions and are stocky principally because they are short in stature. The nine samples deficient in mean weight by $> 1\sigma$ represent the most linear of the American aborigines. Two are the rather tall Alaskan Eskimo samples, and the one from St. Lawrence Island is descended from Asiatic Eskimo migrants with strong physical relationships to the rather tall and linear Chukchi. The Zuni and one of the Navaho samples from the American Southwest are also linear, but may reflect only a temporary dietary linearity. The five remaining linear samples are all Nahua-speakers from the highlands and lowlands of Central Mexico, and probably represent a linearity of a hereditary nature.

On these bases, the factors other than environmental temperatures causing alterations in human physique may be nutritional and non-genic, as in the postulated dietary linearity of the Zuni and one Navaho sample and the obesity attendant upon acculturation in the Papago. Oher factors may be nutritional and largely genic, as seen in the apparent long-term adaptation of small body mass in the highland peoples of Middle and South America. Other trends, such as the tall stature and consequently large body mass of Indians in the U. S. and Northern Mexico, the linearity of the Nahua-speakers of Central Mexico, and the short stature and lateral body builds of the South American lowland Indians, are not immediately explicable, and may represent the combined effects

of hereditary and environmental factors not directly related to environmental temperatures.

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LITERATURE CITED

ANDERSON, R. K., J. CALVO, W. D. ROBINSON, G. SERRANO, AND G. C. PAYNE 1948 Nutritional appraisals in Mexico. Am. J. Public Health, *38*: 1126-1135.

BAKER, P. T. 1958 The biological adaptation of man to hot deserts. Amer. Naturalist, *92*: 337-357.

BASAURI, C. 1929 Mongrafía de los Tarahumaras. Talleres Gráficos de la Nación, México.

——— 1931 Tojolabales, Tzeltales y Mayas. México.

BASAURI, M. no date Ensayo telegético sobre la téchnica agricola de los campesinos de Ocotepec, Mor. (Study presented at the 7th Interamerican Scientific Congress, Washington) Unpublished manuscript.

BASTOS D'AVILA, J. 1950 Anthropometry of the Indians of Brazil. Handbook of South American Indians, *6*: 71-84.

BEALS, R. L., AND E. HATCHER 1943 Diet of a Tarascan town. Amér. Indígena, *3*: 295-304.

BOAS, F. 1901 The Eskimo of Baffin Land and Hudson Bay. Bull. Am. Mus. Nat. Hist., *15*: 1-370.

BULLERUD, J., J. EDWARDS, AND R. A. BLAKELEY 1950 A survey of the basal metabolism of Eskimos. Proj. 21-01-020, Arctic Aeromed. Lab., Ladd AFB, Alaska.

COLLAZOS, C., H. S. WHITE, R. L. HUENEMANN, E. REH, P. L. WHITE, A. CASTELLANOS, R. BENITES, Y. BRAVO, A. LOO, I. MOSCOSO, C. CÁCERES, AND A. DIESELDORFF 1954 Dietary surveys in Peru. III Chacán and Vicos, rural communities in the Peruvian Andes. J. Am. Dietetic Assoc., *30*: 1222-1230.

COLLINS, H. B., JR 1925 Anthropometric observations on the Choctaw. Am. J. Phys. Anthrop., *8*: 425-436.

COMAS, J. 1943 La asistencia pública y el desarrollo biológico del indígena. Amér. Indígena, *3*: 337-344.

——— 1944 Contribución al estudio antropométrico de los indios Triques de Oaxaca. Anales Inst. Etnog. Amér., Univ. Nac. Cuzco, *5*.

CRILE, G. W. AND D. P. QUIRING 1939a Indian and Eskimo metabolisms. J. Nutrit., *18*: 361-368.

—— 1939b A study of the metabolism of the Maya Quiché Indian. J. Nutrit., *18*: 369-374.

D'Aloja, A. 1939 Sobre la variabilidad de algunos caracteres antropométricos observados en grupos de indígenas Centroamericanos. Inst. Panamer. Geog. e Hist., *43*.

Darby, W. J., *et al.* 1956 A study of the dietary background and nutriture of the Navajo Indian. J. Nutrit., *60*: suppl. 2.

Droogleever Fortuyn, A. B. 1946 Some data on the physical anthropology of the Oajana Indians. Konik. Vereenigung Indisch Inst., *59*: 1-24.

Eggan, F. and M. Pijoan 1943 Some problems in the study of food and nutrition. Amér. Indígena, *3*: 9-22.

Environmental Protection Research Division 1958a Canal Zone Analogs I. Analogs of Canal Zone climate in Middle America. Tech. Rep. EP-87, Quartermaster Research and Eingineering Center, Natick, Mass.

—— 1958b Canal Zone Analogs VI. Analogs of Canal Zone climate in South America. Tech. Rep. EP-97, Quartermaster Research and Engineering Center, Natick, Mass.

—— 1959 Climatic analogs of Fort Greely, Alaska and Fort Churchill, Canada in North America. Tech. Rep. EP-111, Quartermaster Research and Engineering Center, Natick, Mass.

Ezekiel, M. 1941 Methods of Correlation Analysis. John Wiley and Sons, London.

Fabricius-Hansen, V. 1949 Anthropological measurements of Greenlanders in the Southern District of Julianehaab. Acta Gen. Statistica Med., *1*: 252-266.

Faulhaber, J. 1950-56 Antropología Física de Veracruz. Gobierno de Veracruz.

Field, H. 1954 Los indios de Tepoztlán (México). Univ. Miami Press, Coral Gables, Florida.

Flores, M. and E. Reh 1955a Estudios de hábitos dietéticos en poblaciones de Guatemala. I. Magdalena Milpas Altas. Bol. Of. San. Panamericana, Suppl. 2, 901-128.

—— 1955b Estudios de hábitos dietéticos en poblaciones de Guatemala. II. Santo Domingo Xenacoj. Bol. Of. San. Panamericana, Suppl. 2, 129-148.

—— 1955c Estudios de hábitos dietéticos en poblaciones de Guatemala. III. San Antonio Aguas Calientes y su Aldea, San Andres Ceballos. Bol. Of. San. Panamericana, Suppl. 2, 149-162.

—— 1955d Estudios de hábitos dietéticos en poblaciones de Guatemala. IV. Santa María Cauque. Bol. Of. San. Panamericana, Suppl. 2, 163-173.

Fuente, J. de la 1945 Sobre nutrición y enfermedades de Indios. Amér. Indígena, *5*: 235-239.

Gabel, N. E. 1949 A comparative racial study of the Papago. Univ. New Mexico Publications in Anthrop., *4*.

Gessain, R. 1958 Les Eskimo d'Angmassalik. Principaux charactères anthropologiques. L'Anthropologie, *62*: 452-484.

Gómez Robleda, J. 1943 Pescadores y Campesinos Tarascos. Ed. de la Soc. de Ed. Pública, México.

GOULD, B. A. 1869 Investigations in the Military and Anthropological Statistics of American soldiers. Hurd and Houghton, New York.

GOULD, H. N. 1946 Anthropometry of the Chol Indians of Chiapas, México. Middle Am. Res. Rec., *1*: 91-110.

HOOTON, E. A. *et al.* 1951 Handbook of body types in the United States Army (White males). Prepared for the Office of the Quartermaster General.

HURTADO, A. 1932 Respiratory adaptation in the Indian natives of the Peruvian Andes. Am. J. Phys. Anthrop., *17*: 137-165.

INCAP 1953 Recomendaciones nutricionales para los poblaciones de Centro América y Panamá. Bol. de la Of. San. Panamericana, Suppl. no. *1*: 119-128.

——— 1955 Recomendaciones nutricionales diarias para las poblaciones de Centro América y Panamá (Revisadas, septiembre de 1954). Bol. de la Of. San. Panamericana, Suppl. no. *2*: 225-226.

JENNESS, D. 1923 Physical characteristics of the Copper Eskimo. Report of the Canadian Arctic Expedition 1913-18. F. A. Acland, Ottawa.

LAUGHLIN, W. S. 1951 The Alaskan gateway viewed from the Aleutian Islands. Pp. 98-126 in Papers on the Physical Anthropology of the American Indian (W. S. Laughlin, ed.). The Viking Fund, Inc., N. Y.

LECHE, S. M. 1936a Dermatoglyphics and functional lateral dominance in Mexican Indians. IV Chamulas. Anthropometry of the Chamulas. Middle Am. Research Series, 7: 289-310.

——— 1936b Dermatoglyphics and functional lateral dominance in Mexican Indians. III Zapotecas and Mixtecas. Anthropometry of the Zapotecas and Mixtecas. Middle Am. Research Series, 7: 229-277.

LECHE, S. M., H. N. GOULD, AND D. THARP 1944 Dermatoglyphics and functional lateral dominance in Mexican Indians. V. The Zinacantecs, Huixteca, Amatenangos, and Finca Tzeltals. With the anthropometry of these four groups. Middle Am. Research Records, *1*: 21-64.

LEVI-STRAUS, C. 1948 The tribes of the Upper Xingú river. Handbook of South Americon Indians, *3*: 321-348.

LUTHER, M. no date Race mixture in Southwest Greenland. Unpublished manuscript deposited in the library of the Peabody Museum, Harvard University.

MARTIN, W. E. 1953 Basic body measurements of school age children. U. S. Dept. of Health, Education, and Welfare.

MONGE, C. 1953 Biological basis of human behavior. Pp. 127-144 in Anthropology Today (A. L. Kroeber, ed.), Univ. of Chicago Press.

NEWMAN, M. T. 1957 The physique of the Seneca Indians of western New York State. J. Wash. Acad. Sci., *47*: 357-362.

NEWMAN, M. T., AND C. COLLAZOS 1957 Growth and skeletal maturation in malnourished Indian boys from the Peruvian Sierra (abstract). Am. J. Phys. Anthrop., *15*: 431.

NEWMAN, M. T., C. COLLAZOS, F. H. ALLEN, H. MARTÍNEZ, AND C. CÁCERES no date An integrated biological and nutritional study of the Quechua Indian population of Vicos, North Peruvian Sierra. Unpublished manuscript.

NEWMAN, M. T., AND C. R. JONES no date The human biology of the Wai-Wai Indians of British Guiana. Unpublished manuscript.

NEWMAN, R. W. 1955 The relation of climate and body composition in young American males (abstract). Am. J. Phys. Anthrop., *13*: 386-387.

NEWMAN, R. W. AND E. MUNRO 1955 The relation of climate and body size in U. S. males. Am. J. Phys. Anthrop., *13*: 1-17.

PAYNE, E. H., L. GONZALES-MUGABURU, AND E. M. SCHLEICHER 1956 An intestinal parasite survey in the high cordilleras of Peru. Am. J. Trop. Med. and Hyg., *5*: 696-698.

PI-SUÑER, B. J. 1933 Studies in racial metabolism. Basal metabolism of the Araucanian Mapuches. Am. J. Physiol., *105*: 383-388.

RANKE, K. E. 1910 Anthropologische Beobachtungen aus Zentralbrasilien. Abh. der Mathe-Physikalischen Klasse der Königlich Bayerischen Akad. der Wissenschaften, *24*: 1-148.

RAY, P. H. 1885 Ethnographic sketches of the natives. Report of the International Polar Expedition to Point Barrow, Alaska, Washington.

ROBERTS, D. F. 1953 Body weight, races, and climate. Am. J. Phys. Anthrop., *11*: 533-558.

ROBERTS, D. F., AND M. T. NEWMAN no date Physiological determinations on Middle American Indians. Manuscript.

RODAHL, K. AND J. EDWARDS 1952 The body surface area of Eskimo as determined by the linear and the height-weight formulas. Proj. 22-1301-0001, Part 1, Arctic Aeromed. Lab., Ladd Air Force Base.

ROMERO, J. no date Estudio antropológico de los Mixtecos de Tilantongo, Oaxaca. Unpublished manuscript.

SCHRAER, H., AND M. T. NEWMAN 1958 Quantitative roentgenography of skeletal mineralization in malnourished Quechua Indian boys. Science, *128*: (33222) 476-477.

SCRIMSHAW, N. S., M. A. GUZMAN, AND O. B. TANDON 1955a Efecto de la aureomicina y penicilina sobre el crecimiento de escolares Guatemaltecos. Bol. de la Of. San. Panamericana, Suppl. no. *2*: 19.

SCRIMSHAW, N. S., M. BEHAR, C. PÉREZ, AND F. VITEN 1955b Nutritional problems of children in Central America. Pediatrics, *16*: 378-397.

SELTZER, C. C. 1936 Physical characteristics of the Yaqui Indians. Pp. 91-113 in Studies of the Yaqui Indians of Sonora, Mexico. Texas Tech. Coll. Bull., *12*: (1).

SKELLER, E. 1954 Anthropological and ophthalmological studies on the Angmagssalik Eskimos. Middelelsen om Grønland, *107*: (4).

STEGGERDA, M. 1932 Anthropometry of adult Maya Indians. Carnegie Inst. Wash., 113 pp.

—— 1941 Maya Indians of Yucatan. Pub. no. 531, Carnegie Inst. Wash.

STEWART, T. D. no date Anthropometric measurements on Highland Maya Indians in Guatemala. Unpublished data.

U. S. WEATHER BUREAU 1954 Monthly climatic data for the world, January, 1954. U. S. Dept. Commerce, 7: (1).

VAVICH, M. G., A. R. KEMMERER, AND J. S. HIRSCH 1954 The nutritional status of Papago Indian children. J. Nutrit., *54*: 121-132.

WILLIAMS, G. D. 1931 Maya-Spanish crosses in Yucatan. Papers Peabody Mus., *13*: (1).

BOOK REVIEWS

Nature and Man's Fate. By GARRETT HARDIN. 375 pp. Rhinehart, New York, 1959. $6.00.

To G. B. Shaw, the professions were conspiracies. Views almost as impish are held by some intellectuals. Hardin, for instance, believes that professional book reviewers are notoriously devoid of understanding (p. 126). We feel that a touch of professional bias may be at times indispensable, as it provides the necessary color and flavor. It is true, though, that professionals harassed for time favor the pick-as-you-go method of reviewing: in a short hour of perusal (by a "professional" worth his salt) most any book will yield enough objectionable passages to concoct a sharp commentary. In this case, reading *Nature and Man's Fate* was no problem. Its style is so fluent, and the lure of its pages so strong that one turns willingly from browsing to reading: just as in Galbraith (the economist), almost every page carries some witticism.

Conway Zirkle, who reviewed Hardin's book for *Isis*, characterized its author's approach as historical, his language unambiguous, and the text exceptionally refreshing. In *Science*, Berrill—while making the point that McGill is in Montreal and not in Toronto (p. 163)—emphasized that the book is excellent ("it could hardly be better") as a lively and informative presentation of the nature and importance of genes for the present and future well-being of man. In *Evolution*, it is called a "lucid account of current evolutionary thought . . . for the intelligent layman." To Ashley Montagu (on the cover flap), the book appeared to be one of the best of its kind. There is no redundancy in these superlatives. Your reviewer loaned his copy of the book to an impartial physician-friend who, too, found it to be as brilliant as a *concert-étude.* In fact, it contains an (over-)abundance of *bon mots—e. g.*, "Nothing is so contagious as ideas; and no contagion is so much altered in the transmission" (p. 216)—and at times it looks as if the author had combed more than the usually alotted quota of dictionaries of citations. An acrimonious reviewer once attributed to the *Vestiges* of Chambers all the attributes of an accomplished harlot (p. 32): whatever those attributes were, Hardin's creation undoubtedly exhibits grace, poise, appeal, and occasional saucy remarks (p. 169, p. 219).

Garrett James Hardin was born in 1915 in Dallas. With a B. S. from

the University of Chicago (1936) he went as a microbiologist to Stanford, where he obtained his Ph. D. (in biology) in 1941. He worked for some time as a plant biologist, then moved to Santa Barbara College (University of California), and began climbing the academic stepladder in the Department of Biology. After two dozen articles, and a successful (1949) textbook on general biology (ed. 2: 1952), he became a full professor. The book presently under review is the palpable result of a sabbatical leave, spent largely at the California Institute of Technology, and at the Huntington Library. The text portrays its author as a refined agnostic (p. 260), and articulate scientific purist, shaped in many ways after his endeared Victorian models.

Veterinary undergraduates enjoy being told that human medicine is but an overgrown branch of veterinary science. Since medicine (either veterinary or human) may be regarded as a branch of biology, some biologists have exulted in dabbling with matters medical. Quite recently, for example, Julian Huxley speculated on the dire (political!) consequences of the alleged overconsumption of tranquilizers in the Western World. *De omni re scibili . . .* , the proud motto of the Renaissance humanist Giovanni Pico della Mirandola, which covered all things that could (ever?) be known, was supplemented—centuries later—with Voltaire's biting . . . *et quibusdam aliis* (and several others). The times of Renaissance humanism are gone, but a few epigonous humanists have disguised themselves as learned biologists. Unfortunate as it may be, medicine has not yet become a science—it is still an art and within its territory, clinical judgment is both the beginning and the end of all theories and interpretations. Hardin acknowledged the dangers which derive from (unverified) " logical thinking " (p. 168) but, lacking personal clinical experience, in medical matters he had to rely on bibliographic lore and on logic, both of which are very desirable assets—though unreliable, unless tempered with actual observations. From this vantage point, it becomes understandable why the weakest parts in Hardin's book are those which contain " para-medical " comments, such as the digression on the " bio-chemic personality " (p. 196). The various difficulties connected with artificial insemination are quite accurately summarized (p. 169) while the blunder comes in the attempt of " psychologic " reconciliation of the sterile husband with the idea that the " semi-adopted " progeny of the wife (sired by a donor) is also " his " child (p. 173). Hardin hypothesizes that the small actual size of the spermatozoid is a valid argument against using " emotionally charged possessive pronouns " (p. 176), *e. g.*, my own child. Has he forgotten that the

"genetic" particles in our gonads are the only potentially immortal fractions of our otherwise so mortal bodies?

What pearls of sarcasm would Hardin muster for the foolhardy physician who were to employ hearsay and reasoning as a basis for dogmatic statements on weed control in aquatic media, or on the alleged damage to crops inflicted by industrialization? And yet, Hardin takes strong issue with an anonymous medical source (dated 1950) which is quoted as denying the existence of concrete evidence of genetic hazards from radiation. To him, this denial appears as outrageous as if a "biologist" were to claim that a free fall from 42,000 feet on "jagged rocks" might not be deadly because there were no "experimental evidence" to this effect (p. 202). The comparison is not only puerile, it is inaccurate. But since the radiation scare is a matter of actuality, it deserves some background information (most of which is not included in Hardin's text).

In 1907 a physician who taught anatomy in the University of Wisconsin, Charles Russel Bardeen (1871-1935), was the first to demonstrate that roentgen irradiation (of toad spermatozoa) may cause undesirable effects in the offspring. In 1927 incontrovertible evidence of roentgen-induced mutations in the fruitfly was adduced by Muller (this *is* in the book), who at the time was a zoologist at the University of Texas. Since 1929 there exists a National Committee on Radiation Protection, but the general public became aware of the problem only in the 1950's, when various pressure groups demanded that testing of atomic weapons be discontinued, to avoid potential genetic (and dosage-wise even less likely somatic) damage from radioactive fallout. To this clamor "conservative" physicists retorted that medical radiation sources contribute a larger (estimated) average total body dose than present-day fallout. While this did not solve the dilemma "to test or not to test" it distributed the overall pressure more evenly by co-involving in the controversy physicians in general, and radiologists in particular.

The "geneticists" contend that genetic radiation hazards to humans are sufficiently proved by experimental results obtained in fast reproducing insects and small mammals. This has led to often daring, and sometimes hysterical, extrapolations. Current concepts postulate that absorption of a single quantum of ionizing radiation will cause at least some cell damage which, in the gonads, might well be a mutation. This experimental finding speaks for itself (it has never been denied in medical circles) but, with Candide, let us look at the clinical facts. Diagnostic roentgenology has been in existence since 1896, and in current

(often indiscriminate) use for most of the past four decades—yet, so far, there has been no demonstrable rise in obvious mutations. Moreover, it has been stated (if you can believe such computations) that more dangerous than roentgen rays—mutation-wise—is the "civilized" habit of "overheating" the testicles by wearing pants: if the proselytes of Venus Castina find out about that, we may soon be fighting a drive which would have us change to kilts.

In the 1920s Ira Kaplan, a New York radiologist, administered roentgen therapy to several hundred "apparently sterile" women, after all known medical procedures had failed to relieve their infertility. In every one of these patients, each ovary received 225 r(oentgen) in air, which is over 100 r tissue dose (by comparison, LD^{50} for total body radiation in humans is about 500 r). Thereupon, many of the irradiated women bore children, who grew up, married, and had children of their own. Since then, at periodic intervals, Kaplan exhibited photographs of these grandchildren (at the time of his latest publication there were about thirty of them), and *none* had any demonstrable abnormalities. Modern geneticists would have us wait through the eighth generation, to accept it as a proof that there are no latent carriers among these descendants of irradiated ovaries, which may be theoretically correct—but by then all the staunch litigants will at best be memories. Today, roentgen therapy for dysmenorrhea (and infertility) is hopelessly obsolete. Still, let us not forget that these thirty apparently healthy youngsters are with us either or both because and despite the ovarian irradiation inflicted upon their grandmothers.

There is a clinically indubitable somatic hazard connected with ionizing radiation, and therefore neither their promiscuous use, nor inadequate protective measures, may ever be condoned. But there are faint indications that the pendulum of scientific opinion is about to swing back, and allow for less overt anxiety over the genetic hazards of the invisible light. On the other hand, various explanations have been offered (aside from the genetic time-schedule) for the discrepancy between the experimentally demonstrable, but clinically undetectable, radiation-induced mutations. First of all, deaths due to lethal dominants occur too early to be of importance as human hazards. Lethal recessives do not manifest themselves unless and until mating takes place, and only then in certain proportions. As for the (allegedly few) "good" and (allegedly many) "bad" non-lethal mutations, said to be in the majority recessive, one might assume that imperceptible selection would take care of them, as it does take care of the presumably numberless "spontaneous"

(cryptogenetic ?) mutations, known to occur in the absence of measureable ionizing radiation. After all, there are populations living for millenia (on monazite beaches in India, on vulcanic soil in the Andes) in the presence of a comparatively elevated "background" radiation, which did not seem to have impaired them in any demonstrable way. There is no doubt in this reviewer's mind that a few decades hence, some of today's more articulate "genetic radiation damage"-ists will be cited in the same humorous vein as the hazards to health which overcautious forebears ascribed to the 40-50 MPH speed of trains of one century ago.

Let's return now to Hardin's book. Following the prologue and the acknowledgments, there are a total of thirteen chapters, after which come brief notes, 215 references, and a fairly detailed index. Appearing at the sesquicentennium of Darwin's birth, it brings biographies of the *Origin of Species*, of the latter's author, and of other notables of the period. Chapters six through twelve, while interwoven with historic items, constitute a very cursive account of presently available knowledge on evolutionary theory, including—of course—genetics. There occur several premonitory sections—such as the remarkable *Heavy hand of equalitarianism* (p. 189)—but the author's message is concentrated in the last chapter, entitled *Praise of waste* (pp. 300-346), published previously as a separate unit in the *Saturday Evening Post*. This message cannot be summarized, it must be read. Your reviewer naturally disagreed with several of the author's theses, but this made it more interesting: it is undoubtedly immensely stimulating.

Turning now to one of the crucial problems in the book—man's fate—the reviewer feels that this cannot be intelligently discussed until a very old tenet, popularized mainly by Malthus, has been decidedly debunked. This old tenet was recently incorporated in an exhibit (at the Field Museum of Natural History in Chicago) which portended that if a pair of elephants, and their descendants, were to breed without external interference, 750 years later their progeny would number 19,500,000. *This is a strident fallacy!* The reproduction curve of a microbian or tissue culture—given all the food, space, and conditions wanted—will always tend toward saturation. The lack of knowledge regarding the mechanism of this intrinsic phenomenon should not be misconstrued as implying an animistic explanation. Some kind of "finalistic" relationship must exist between the increased loss of males during wartime, and the concomitant rise in the incidence of male births, even though we do not know over what channels this feedback operates. The threat of extinction is what keeps the natality up. Ritualistic slaughtering by primitive

people is likewise a fertility-fostering procedure—and not the result of a threat of overpopulation, as assumed by Hardin (p. 318). When all biologic battles have been won, fertility increases, and this is probably how and why the dinosaurs disappeared. Without grasping this basic fact, it is doubtful whether anything in biology can be adequately interpreted.

What then should be the concluding judgment on Hardin's book? The value of any work can be determined only in a relative way, that is in relationship to a certain individual (or perhaps to a group of individuals). After having read it, in the event of loss of the book, would he re-buy it? The physician-friend, mentioned at the beginning of this review, never returned the original copy loaned to him, and your reviewer —*in extremis*—purchased another copy. Aside from intellectual stimulation, what one can expect to find in Hardin's book is perhaps best expressed in a quotation from Baco de Verulam, which this reviewer copied from (*anathema*!) Booth's *History of Osteopathy*. The quotation applies quite well to the history of evolutionary theories, and in a wider sense to all discoveries in biology. It reads: "He who will not reason is a bigot; he who can not reason is a fool; and he who dares not reason is a slave."

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Hair Growth and Hair Regeneration. IRWIN I. LUBOWE (ed.) Annals of the New York Academy of Sciences, Vol. 83, pp. 359-512, 1959.

Ten years ago a reference-shelf on hair required little space, a few linear feet sufficing for the Pincus and Friedenthal monographs, Danforth's *Hair*, reprints of Hamilton, Hausman and Trotter and even semi-scientific books like Goodman's and Savil's. There was room for Sorby's researches (from the Journal of the Royal Microscopical Society), Metchnikof's marvelous melanophages and observations on kemp, felting-fur and *Lagothrix*. Now, in a decade, the hair-shelf has quadrupled, and no one person can encompass all of the directions of active hair research which procede in both depth and breadth.

Certain problems continue to be of interest, especially male pattern balding, and J. B. Hamilton's earlier observations do hold. Neither

heat nor hats nor loss of fat account for male patterned alopecia, but rather factors intrinsic to the hair root (presumably set down before birth) in the presence of particular androgenic steroids. The problem of neoformation of hair follicles continues, with the better evidence still against mass neoformation, excepting perhaps in ovarian and dermoid cysts.

There is little stress in this decade on medullary counts and cuticular patterns but renewed interest in the century-old layers of Henle and Huxley. Through chromatography, isotope studies, x-ray diffraction and histochemistry, the constituents of the growing hair are becoming known. Electron microscopy adds to description at a micro-micro level, while investigations on cutaneous enervation promise to explain waves of growth and (ultimately) non-patterned alopecias.

As always, investigation bells the cat of speculation. Following thallium ingestion there is no lack of keratinization, rather a super-keratinization at the level of the root sheath. What is of interest throughout this volume (unlike the *Growth, Types and Replacement of Hair* of a decade ago) is the extent to which biochemical approaches now pervade all aspects of hair research, even those most classically anatomical. Under the microscope the hair root merely simulates a less-pigmented shaft. Adding phase -microscopy or polarization techniques, the lesser keratinization of the bulb is obvious, but chemical characterization is still lacking. Thus the compleat hair-man, adding this monograph to his shelf, must of necessity plan biochemical and biophysical additions for his toolkit of investigative techniques.

This conference report, which represents work in progress, joins Rothman's *Physiology and Biochemistry of the Skin* and Montagna's *The Biology of Hair Growth* in producing a comprehensive picture of hair, its composition, growth and replacement.

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Build and Blood Pressure Study. Volume 1. 268 pp. Society of Actuaries, Chicago, Ill., 1959. No price indicated.

Large intercompany mortality investigations are undertaken periodically for underwriting purposes: In what ways do various characteristics, ascertainable at the time of the application for life insurance and referred

to technically as "impairments," affect mortality? The answer to this questions aids the underwriter in deciding whether or not to insure a particular applicant, and, if so, at what rate.

Human lives and factors affecting longevity are also the supreme "business" of human biology. Thus it appears proper to consider the volume with reference to physical anthropology, with emphasis on weight and height. The major feature of mortality experience with reference to blood pressure measured at policy issue is the sharp rise in mortality even with relatively small reported elevations in blood pressure. The elevation of the level of the blood pressures in life insurance applications may be understated.

The present investigation covered the intercompany experience under ordinary insurance issued during the years 1935 through 1953, traced to policy anniversary in 1954. Policies issued at ages under 15 or over 69 were excluded as were deaths attributable to war. Annual mortality rates have been decreasing steadily. As an example, for age group 30-34 (at issue) and 6-10 policy years, the annual mortality rate per 1,000 during the periods 1909-27, 1925-37, 1935-50, and 1935-54, were 4.25, 3.88, 2.10, and 2.03 respectively. The last figure is less than one half of the mortality in the first quarter of this century. It would have been valuable to provide parallel data on the mortality of the U. S. White population or its specified segments. Unfortunately, direct comparisons are difficult because the insurance data are tied to "age at issue" and policy years while vital statistics indicate mortality in specified age groups.

An important contribution of the insurance studies is the information they yield on heights and weights. The present study provides data, based on a stratified sample (290,000 policies on men; 70,000 policies on women) of the insured population. Average weights are indicated, for each inch of height, for 8 age groups and each sex. As before, these represent the weights with shoes and indoor clothing.

The 1912 study, in which heights were also obtained with shoes, shows—in contrast to all other studies of which the reviewer is aware—that the average height of 68.5 inches was maintained essentially throughout the whole adult life (25-69 years). It appears to be an inappropriate standard of reference, since it would lead to the unlikely conclusions that in the present study younger people (69.5 inches at 20-29) are taller but older men are shorter (68.1 inches at 60-69) than half a century ago.

Similar, perhaps even greater difficulties are encountered in com-

paring average weights reported in various studies. Adjustments involving adding 1 inch and 8 pounds to data for men, 2 inches and 5 pounds for women in samples measured without shoes and clothing, are not justified by reference to any empirical data. The comparison of the 1912 and the 1959 study (not free from problems of sampling, changes in the weight of clothes and the height of shoes, especially among women) suggests, at a given height, a tendency toward *increased* weight among men, with averages ranging from 1 to 8 pounds at different ages and heights, and average *decreases* of up to 6 pounds among women.

The core of the volume is constituted by the tables relating "build" and blood pressure to overall mortality experience and to mortality experience by cause of death. Although there is a substantial degree of variability in the mortality ratios in the numerous weight classifications by age groups and height, overweight tends to be associated with excess mortality. With few exceptions (ages 15-19 at issue, tall men at issue ages 20-29), the most favorable mortality experience tends to be associated with weights below the average. Among men the range of weights associated with low mortality extends in some instances to the average weight group. For some groups the weight ranges associated with lowest mortality are quite broad; e. g. for men of medium height, 30-39 years of age at issue, the limits are 115 and 164 pounds. Without empirical evidence to back the statement, the idea is set forth that "within any weight range . . . it is likely that the highest weights apply to individuals of large frame and the lowest weights to individuals of small frame" (p. 47).

In regard to mortality experience by cause of death, among overweights of both sexes the increased mortality is accounted for primarily by excess death rates from cardiovascular-renal diseases, diabetes, and diseases of the digestive system. These findings are not new but are based on a larger material than was available prior to the present study.

For the overweight individual the question of "What reward may I expect from reducing" is very real. Some answers are provided by information on the mortality among persons initially overweight who became eligible for standard insurance following reduction in weight. The overall results appear very satisfactory, indeed, with the mortality ratio reduced to 102, i. e. to a level essentially identical with that of the "standard risks." The sample is, unfortunately, small (total N = 225 policies for men), effects of underwriting selection at the time of change to standard issue remain an unknown factor, and strikingly different results are obtained depending on the way the expected mortality

is computed, i. e. with reference to policy duration since *original* issue date or since *change to standard* insurance. For policy duration of 1-5, 6-10, and 11-15 years we obtain mortality ratios of 82, 101 and 106 in the first case, and 85, 108 and 142 in the second case.

The unquestionable advantage of the present study as a whole is the size of the samples: In the build study the total number of "entrants" (life insurance policies) approximated 5,000,000, the average duration of exposure (follow-up period) was close to 8 years, and the total number of policies terminated by death was 133,000.

Although these numbers are impressive, occasionally mortality in specified subgroups is based on distressingly low values of "N." Thus the finding that women with rapid pulse have an excess mortality of 15%, as compared to corresponding female risks without known impairments, are based on 11 policies terminated by death (p. 179). Among men with family history of cardiovascular-renal disease, the mortality from diseases of the heart and circulatory system rises to 4 times the expected for entrants with elevated diastolic blood pressure (93-102 mm Hg) but this finding is based on only 13 policies terminated by death. These are extreme examples, but they bring out two related points: 1) It is in these small subgroups that the findings may be readily affected by the fact that the mortality experience was studied in terms of policies rather than individuals; a single individual having a number of insurance policies may profoundly influence the results. 2) The dependability of the ratios of actual to expected mortality decreases as the number of deaths upon which it is based diminishes, and even in insurance statistics one must be concerned about the confidence limits of the mortality ratios. The underlying concepts were clarified (pp. 14-15) and tables were prepared (pp. 242-245) showing lower and upper limits, as deviations from the observed mortality ratio, at the 50% and the 95% confidence level for samples of 35 or less.

Important features of the study and some of its major limitations are consequences of the fact that it is the byproduct of a commercial enterprise, not the result of an investigation designed for strictly scientific purposes. To begin with, "build" refers to variations in height and to variations in weight at specified heights. This is, of course, a good deal simpler definition of body build than the physical anthropologist is ready to accept. He would wish to emphasize the need for taking into account the lateral dimensions of the bony framework in arriving at "standard" weight, a point to which lip service was paid by the insurance industry in the tables of "desirable weight" without offering any objective criteria

for the classification of skeletal frames into "small," "medium," and "large."

While there is a significant degree of association between underweight-overweight and "leanness-fatness," so that *groups* of overweight subjects are also over-fat, there are *individuals* who are heavy but "lean"; among middle-aged men those physically more active tend to differ from those who are physically less active primarily in terms of the fat-free weight. These distinctions are beyond the reach of the present *armamentarium* of physicians making physical examinations for the life insurance industry but the distinctions have biologically significant relationships to morbidity and mortality.

The authors, in general, spell out the limitations both in regard to the basic biological information ("Nature of Recorded Weights and Heights," p. 24-25; "Nature of Recorded Blood Pressure Readings," p. 36-37) and the conclusions that can be drawn from the ratios of actual to expected mortality in specific classifications according to height, weight and blood pressure: "These mortality ratios—while highly significant for life insurance underwriting, are not always so meaningful for clinical practice or public health purposes" (p. 44).

The basic standard of reference is the mortality experience among persons insured under ordinary life insurance policies issued at standard premium rates. The Basic Table (p. 7) represents combined experience on male and female lives. Because of the important sex differences in mortality, the table provides "only an approximate measure of the mortality of men . . . and overstates greatly the mortality of women who qualify for standard life insurance" (p. 44).

The sex differences in mortality pose intriguing questions. These involve not only the lower level of mortality women, but also the role of genetic factors. Men with a family history of cardiovascular-renal diseases have an elevated death rate, with an overall mortality ratio of 144%. Among women no such excess mortality is present. This is a striking difference, even though the female sample was small. The differences in the relative role of diseases of the cardiovascular system and of cancer are clear cut. Typically, for ages 30-39 at issue and 6-10 policy years, among men the two categories of disease account for 47.5 and 16.1% of all the deaths, while in women the figures are 13.7 and 37.1%, respectively; *i. e.*, the relative magnitudes are reversed.

The interpretations are, in general, cautious and sound. The treatment of height is an exception. For men, the maximum height was observed in the age group 20-29, the minimum at 60-69; the actual

means were 69.5 and 68.1 inches, yielding a difference of 1.4 inch. For women the corresponding values were 64.7 and 64.2, a difference of 0.5 inch. The observed differences were interpreted not as a partial evidence of an increased stature of the American population but as a direct measure of it: "The changes in average height with age in the present study suggest that over a period of some 40 years there has been an increase of nearly 1½ inches in height among men but only about one-half inch among women" (p. 21). The fact is, of course, that the observed age decrements are complex data which, in part, reflect a true decrease in stature with age and, in part, may reflect a generational increase in stature.

There are signs pointing to developments in the life insurance practices in two directions, one being of social, the other of biological importance. On the one hand, with an ever wider segment of the population being covered by life insurance and various types of life insurance being made available on a group basis, more and more policies are written without a medical examination of the applicant. Under these circumstances the particular group (or the policyholders of a company as a whole) share the risk of the death of the individual members as is the established practice in the health insurance field in regard to illness. On the other hand, the basic problems of social and personal (biological, and perhaps also psychological) factors in longevity and their scientific study are receiving increased attention. The potential contribution of the insurance industry is large, not only because of the financial support that can be devoted to such investigative work but also on account of the facilities for selecting, describing and following up well-defined population samples.

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BOOK NOTICES

GENETICS AND TWENTIETH CENTURY DARWINISM. Cold Spring Harbor Symposia on Quantitative Biology, Volume 24. xv + 321 pp. Biological Laboratory, Cold Spring Harbor, New York, 1959. $8.00. Focused on evolutionary processes related to genetics, without neglecting the effects of the environment, studied by ecologists, and the evidence contributed by anthropologists and paleontologists. It is fitting that this outstanding symposium was held in Cold Spring Harbor where, in 1904, the first laboratory devoted to the study of Mendelian inheritance was established under the name of "Station for Experimental Evolution."

AN INTRODUCTION TO ANTHROPOLOGY. 2d. Ed. By Ralph L. Beals and Harry Hoijer. xxi + 711 pp. Macmillan, New York, 1959. $6.90. An elementary textbook, organized around two major themes: man as a biological organism (pp. 22-222), and the structure and development of culture. In the field of physical anthropology emphasis is placed on the genetic approach (see esp. Ch. 4, "Heredity and Genetics," and Ch. 7 "Race, Evolution and Genetics.").

REFLEXES TO INTELLIGENCE. Edited by Samuel J. Beck and Herman B. Molish. xiv + 669 pp. The Free Press, Glencoe, Illinois, 1959. $8.50. The volume, with a subtitle "A Reader in Clinical Psychology," offers a wide selection of writings (73 articles) contributing to the growing understanding of man's behavior.

EXPERIMENTS IN HEARING. By Georg von Békésy. Translated and edited by E. G. Wever. x + 745 pp. McGraw-Hill, New York, 1960. $25.00. A unique record of investigations on hearing, extended over a period of three and half decades. The four main sections deal with the anatomy of the ear, the conductive processes (including, importantly, bone conduction), the psychology of hearing, and the cochlear mechanics.

HANDBOOK OF AGING AND THE INDIVIDUAL: PSYCHOLOGICAL AND BIOLOGICAL ASPECTS. Edited by James E. Birren. xii + 939 pp. University of Chicago Press, 1959. $12.50. A collaborative, technical summary of the literature on changes in behavior and capacities of the individual organism which occur with advancing age. Biological aspects are examined in detail in Section 2 (pp. 119-278), including problems of growth and aging, mortality of populations, and genetics of individual differences in aging. Relevant topics, such as bodily changes with age or formal models of physiological aging, with application to human mortality data, are discussed in the other three sections (Foundations of Research on Aging, Aging in Environmental Settings, and Psychological Characteristics of Aging).

EFFICIENCY OF HUMAN MOVEMENT. By Marion R. Broer. 351 pp. Saunders, Philadelphia, 1960. $6.00. The core of the book is in the section on the application of mechanical principles to fundamental physical skills, such

as walking, lifting or carrying. Its goal is to provide foundation for physical education.

CONTRIBUTIONS TO THE PHYSICAL ANTHROPOLOGY OF THE SOVIET UNION. By V. V. Bunak, G. F. Debets and M. G. Levin. vii + 192 pp., Volume 1, No. 2, Russian Translation Series of the Peabody Museum of Archaeology and Ethnology, Harvard University. Peabody Museum, Cambridge, Mass., 1960. $4.50. Except for a general methodological paper by Bunak, concerned with "descriptive" (qualitative) characters, the volume presents recent work on the anthropometry and paleoanthropology of the Soviet Union, especially the Caucasus.

THE ANTECEDENTS OF MAN. By W. E. Le Gros Clark. vii + 374 pp. Quadrangle Books, Chicago, 1960. $6.00. The subtitle "An Introduction to the Evolution of Primates" indicates the pitch of this dependable presentation which grew out of a series of lectures on "The Paleontology of the Primates and the Problem of Human Ancestry."

AN INTRODUCTION TO SOCIAL BIOLOGY. By Alan Dale. vii + 434 pp. C. C. Thomas, Springfield, Illinois, 1960. $6.50. A reprint of the third edition, with revisions, of an interesting presentation of "applied human biology." Has a chapter on "Population."

DISEASES OF METABOLISM. 4th Ed. Edited by Garfield G. Duncan. xxii + 1104 pp. Saunders, Philadelphia, 1959. No price indicated. In this, by now classical, treatise of special interest are the discussions of changes in body composition in undernutrition (A. Keys), some parts of the chapter on obesity (J. M. Strang), and the appendix compiled by the editors and presenting height-weight data on children and adults, the latter going back to the 1912 Medico-Actuarial Mortality Investigation. One wonders what is the evidence for allowing 10 lb. for the clothes of men, 6 lb. for the clothes of women.

FALLOUT: A STUDY OF SUPERBOMBS, STRONTIUM 90 AND SURVIVAL. Edited by John M. Fowler. 235 pp. Basic Books, New York, 1960. $5.50. In Adlai E. Stevenson's introductory words, "This is not a book merely about fallout. It is a book about our nuclear quandary—about circumstances that make our period the most uneasy in the history of mankind." W. R. Guild's chapter on "Biological Effects of Radiation" and J. F. Crow's "Radiation and Future Generations" are closest to the biologist's professional concerns.

DEVELOPMENTAL PSYCHOLOGY: AN INTRODUCTION TO THE STUDY OF HUMAN BEHAVIOR. 3d. Ed. By Florence L. Goodenough and Leona E. Tyler. xviii + 552 pp. Appleton-Century-Crofts, New York, 1959. $6.00. Time represents an important parameter of behavior and a good purpose is served by looking upon the human life as a process in which no single phase can be properly considered by itself. While the emphasis is on child development, 3 chapters are given to maturity and old age.

DEATH OF ADAM. By John C. Greene. 388 pp. Iowa State College Press, Ames, Iowa, 1959. $4.95. A historian's acount of "the rise of evolutionary views of nature and the decline of static creationism in the two centuries separating Isaac Newton and Charles Darwin." Richly illustrated and documented.

HUMAN BIOCHEMICAL GENETICS. By H. Harris. viii + 310 pp. Cambridge University Press, New York, 1959. $7.00. An authoritative survey or research on inborn differences between human beings and, more specifically, on those differences that can be formulated in biochemical terms (amino acid and carbohydrate metabolism, hemoglobins, blood-group substances, plasma proteins). Significantly, the subtitle of A. E. Garrod's paper (1902) which initiated the scientific study of biochemical genetics was "A study in chemical individuality." The concluding chapter concerns gene action.

A TEXTBOOK OF HUMAN EMBRYOLOGY. By R. G. Harrison. xi + 244 pp. C. C. Thomas, Springfield, Illinois, 1960. $10.50. Designed "to facilitate the comprehension of human development (prenatal and postnatal) . . . which can, in turn, assist understanding of human anatomy," with accent placed on function.

FUNCTIONAL ANATOMY OF THE LIMBS AND BACK. 2nd Ed. By W. Henry Hollinshead. viii + 403 pp. Saunders, Philadelphia, 1960. $9.00. The subtitle clearly specifies the purpose of the book: A Text for Students of Physical Therapy and Others Interested in the Locomotor Apparatus.

MANKIND IN THE MAKING: THE STORY OF HUMAN EVOLUTION. By William Howells. 382 pp. Doubleday, New York, 1959. $4.95. This is *Mankind So Far*, a presentation combining in an outstanding manner reliability with readability, only *further*: "This is not a revised version, rather, it is a new book by a revised author."

PATIENTS, PHYSICIANS AND ILLNESS: SOURCEBOOK IN BEHAVIORAL SCIENCE AND MEDICINE. Edited by E. Gartly Jaco. viii + 600 pp. The Free Press, Glencoe, Illinois, 1958. $7.50. The 55 chapters, of which about one-third represents new material, deal with various facets of an area so new that we lack a proper label for it. Cultural anthropologists and social psychologists would resent losing their professional identity in "medical sociology," a term that is at times used in the absence of a more comprehensive designation. Most relevant to human biology are probably the papers in the section "Social and Personal Components of Illness."

ANATOMY AND PHYSIOLOGY OF SPEECH. By Harold M. Kaplan. ix + 365 pp. McGraw-Hill, New York, 1960. $6.50. A textbook of physiology organized around the speech mechanisms (the air blast generated by the respiratory system, the structures for phonation and articulation, and the auditory mechanisms for sound reception).

QUANTITATIVE METHODS IN PSYCHOLOGY. By Don Lewis. xii + 558 pp. McGraw-Hill, New York, 1960. $9.50. Describes the uses of mathematics and statistics in the framework of psychological experimentation and theorizing.

BIOLOGICAL PSYCHIATRY. Edited by Jules H. Masserman. xvi + 338 pp. Grune and Stratton, New York, 1959. $9.75. Proceedings of the 1958 scientific sessions of the Society of Biological Psychiatry. The Society was organized in 1945 as a meeting ground for individuals who are interested in the biological basis of behavior and feel the need for integrative thinking in this complex field. The present volume stresses heavily the contributions of psychopharmacology.

MENTAL RETARDATION: ITS CARE, TREATMENT AND PHYSIOLOGICAL BASE. By Hans Mautner. vii + 280 pp. Pergamon Press, New York, 1959. $5.50. An

introduction to "oliogophrenology," in the form and style of a set of lectures. A lecture is devoted to "Genetically Determined Diseases," to "Phenylpyruvic Oligophrenia and Other Inborn Errors of Metabolism," and to "Thyroid Diseases."

HUMAN HEREDITY. By Ashley Montagu. 397 pp. The World Publishing Co., Cleveland, Ohio, 1959. $5.00. A popular survey, emphasizing the interaction between the genetic and the environmental factors, including ionizing radiation. A classified list of inherited disorders is presented in the appendix, together with addresses of heredity clinics and a glossary.

FEDERAL FUNDS FOR SCIENCE. VIII. THE FEDERAL RESEARCH AND DEVELOPMENT BUDGET, FISCAL YEARS 1958, 1959, AND 1960. National Science Foundation. iii + 74 pp. U. S. Govt. Printing Office, Washington, D. C., 1959. 50¢. Continuation of a series of reports begun in 1953. The present issue contains new information concerning the "performers" of the governmentally supported basic research. The percentage distribution for the fiscal year 1959 is as follows: educational institutions, 49%; intramural, 37%; profit organizations, 5%; other, 9%. By field of science, physical, life, and social sciences accounted for 69, 29, and 2% of the total of 488 million dollars, respectively.

PRATIQUE ANTHROPOLOGIQUE. By Georges Oliver. 299 pp. Vigot Frères, Paris, 1960, 32 NF. In the footsteps of Topinard (Eléments d'Anthropologie Générale, 1885), the author presents a manual of techniques of physical anthropology. The first part deals with observations on the living man, with sections on morphological and genetic characteristics (such as blood groups and taste sensibility), while the second part is concerned with the skeleton. Selected references are given in individual chapters and there is a general bibliography at the end of the volume. A short chapter is devoted to statistical techniques.

OUTLINE OF HUMAN GENETICS. By L. S. Penrose. xii + 146 pp. Wiley, New York, 1959. $2.50. An introductory presentation. The concluding chapter is devoted to "Eugenics and Dysgenics."

WORK AND THE HEART. Edited by Francis F. Rosenbaum and Elston L. Belknap. xxii + 537 pp. Hoeber, New York, 1959. $12.00. The topics discussed range from basic physiology to work classification and workmen's compensation. Chapters on occupational and environmental factors involved in the development of heart disease, together with methods for the appraisal of cardiac performance constitute the section of "Clinical Physiology" (pp. 103-229).

BIENNIAL REVIEW OF ANTHROPOLOGY, 1959. Edited by Bernard J. Siegel. vi + 372 pp. Stanford University Press, 1959. $6.00. A welcome addition to tools designed to help anthropologists and members of related specialties keep abreast of the flood of technical literature. "Recent Advances in Physical Anthropology" (pp. 1-36) are described by Gabriel Lasker.

A SHORT HISTORY OF SCIENTIFIC IDEAS TO 1900. By Charles Singer. xvii + 525 pp. Oxford University Press, New York, 1959. $8.00. Written by one of the outstanding historians of science "to give an elementary idea of how science came to occupy its distinctive position in the life of our own time." Biology receives due consideration throughout and occupies a prominent

place in the portrait of the developments during the second half of the 19th century (pp. 461-516), including the rise of genetics.

GENETICS AND CANCER. Edited by the Staff of the University of Texas M. D. Anderson Hospital and Tumor Institute. 459 pp. The University of Texas Press, 1960. $8.50. In this collection, those papers that are likely to appeal most to the readers of this journal have been grouped in the section "Heredity and Human Cancer" (pp. 377-438). They include a presentation of recent developments in human genetics (W. J. Shull), a survey of methods (N. E. Morton) and two "applied" studies (Madge T. Macklin and C. P. Oliver, dealing with breast cancer).

HANDBOOK OF POPULATION CENSUS METHODS. VOLUME III. Statistical Office of the United Nations. vi + 78 pp. United Nations, New York, 1959. 80¢. This final volume, in the series "Studies in Method," deals with the demographic and social characteristics of the population (cf. Irene B. Taeuber's review of Vols. 1 and 2 in *Human Biol., 31*: 360-362).

THE GIFTED GROUP AT MID-LIFE. By Lewis M. Terman and Melita H. Oden. ix + 187 pp. Stanford University Press, 1959. $4.50. This is the fifth volume of the "Genetic Studies of Genius," the most systematic and certainly the most persistent psychobiological study on the record. The present report is based on 35 years' follow-up of a group of superior eleven-year-olds, initiated by Professor Terman in 1921, and provides documentary evidence that "the gifted child is far more apt to become the intellectually superior, vocationally successful, well-adjusted adult than will the average" (p. xii).

ALCOHOLISM: THE NUTRITIONAL APPROACH. By Roger J. Williams. x + 118 pp. The University of Texas Press, 1959. $2.50. The author is convinced that biochemical individuality, i. e. the distinctiveness of the body chemistry, is an important factor in alcoholism. Alcoholism is regarded essentially as a disease of appetite (an excessive appetite for alcohol) and high hopes are held out that it can be prevented by nutritional means.

MODERN NUTRITION IN HEALTH AND DISEASE. 2nd Ed. Edited by Michael G. Wohl and Robert S. Goodhart, 1152 pp. Lea and Febiger, Philadelphia, 1960. $18.60. A broad, authoritative, up-to-date treatment of normal nutrition, nutrition in periods of physiological stress, and in disease. Of special interest to human biologists may be the chapters on "Body Weight, Body Composition and Caloric Status" (A. Keys) and "Obesity" (M. G. Wohl).

SYMPOSIUM ON BASIC RESEARCH. Ed. by Dael Wolfle. xx + 308 pp. Publication No. 56, American Association for the Advancement of Science, Washington, D. C., 1959. $3.00. Sponsored by the nation's top ranking scientific organizations and a foundation known for its support of research based not upon specific "project" proposals but upon the demonstrated capacity of the recipient scientists (Alfred P. Sloan Foundation), the symposium dealt comprehensively with the problems and especially the support of basic research, "the untrammeled search for new knowledge for its own sake" (Warren Weaver, Preface, p. xi).

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